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CLINIC OF DR. ARTHUR DEAN BEVAN[1]

PRESBYTERIAN HOSPITAL

TWO CASES OF GALLSTONE DISEASE

I SHALL present to you this morning two patients who came in with the diagnosis of gallstone disease. The first patient, Mr. T. G., a man of forty-five, has been treated for a number of years for ulcer of the stomach and also for colitis. In going over his history I was not at all satisfied that he had ever had an ulcer of the stomach or of the duodenum. I was convinced that he had had more or less mucous colitis, but this did not explain the entire clinical picture. He gave me quite a definite description of the attack which was sharp and severe, coming on independently of meal taking or of the condition of his colon, lasted for several hours and disappeared under the hot-water bag and occasionally morphine. I, therefore, ordered a flat *x*-ray taken without the Graham test and this showed definitely stones in the gallbladder. Examination of the stomach and duodenum failed to show any evidence of peptic ulcer. I, therefore, advised an operation upon the gallbladder, probably a cholecystectomy.

Dr. Herb anesthetized the patient with the sequence of ethylene and ether. There was no contradiction to ether. I always prefer to use the sequence of ethylene and ether with gallbladder work where there is no definite contraindication.

[1] From October 15–18, 1929, inclusive, I gave four clinics to the Fellows of the American College of Surgeons at their annual meeting in Chicago. These clinics which I am reporting here are a summary of those cases.

I am making the S-shaped incision which we have employed so generally in surgery of the gallbladder and bile tracts. I want to call your attention to the first steps of this incision and

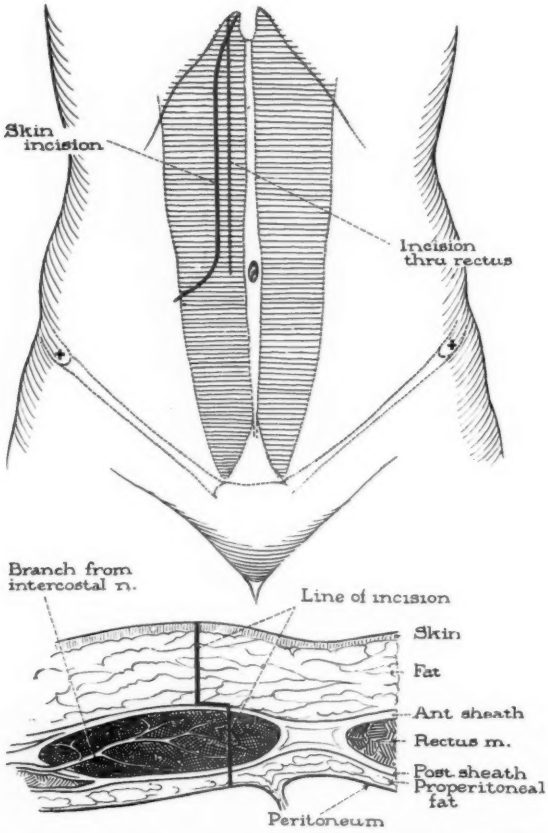

Fig. 83.—S-shaped incision employed in surgery of gallbladder and bile tracts.

to the principles upon which this incision is based. We begin the incision in the angle between the ensiform cartilage and the costal arch (Fig. 83), carrying it down for a distance of about

3 inches parallel with the costal arch and then curve the incision downward over the rectus muscle and at about the junction of the inner fourth with the outer three fourths of the muscle. Then I carry the lower vertical incision downward to a point opposite the umbilicus and then curve the incision outward for about 2 inches. We divide the skin and superficial fascia and the external sheath of the rectus, then split the rectus muscle, leaving about $\frac{1}{3}$ inch of the rectus muscle internal to the incision, and leave the balance of the rectus muscle external to the incision. I regard this as very important, to split the rectus muscle at about the junction of the inner fourth with the outer three fourths because, in the closure, this kind of incision enables us to close the posterior sheath of the rectus in a very satisfactory way with the same sutures which we use in sewing up the peritoneum. This closure of the posterior sheath of the rectus is one of the most important features in preventing a resulting hernia in these rectus incisions. You will remember that the nerve supply of the abdominal wall is furnished from the lower six intercostal nerves. In planning the incision as we are making it now we do not paralyze any of the rectus muscles except the small narrow strip which is left internal to the incision. The rest of the rectus muscle is supplied normally and does not lose its motor nerve supply in any way. The fibers of the rectus are now separated and I divide the posterior sheath and the peritoneum avoiding any injury to the round ligaments which passes from the umbilicus up to the liver beneath the incision. I introduce my hand into the peritoneal cavity and examine the stomach, duodenum, and pylorus and find that they are normal. I find that the liver is normal in appearance.

Examining the gallbladder I find that it is filled with stones. I bring the gallbladder into view and separate the fundus of the gallbladder from the liver. I always begin at the fundus and almost never do a cholecystectomy by first dividing the cystic duct. In an easy case a cholecystectomy can be very rapidly done by beginning with a division of the cystic duct and shelling out the gallbladder from below upward, but in difficult cases, especially with a fixed gallbladder deeply situated and bound

down by adhesions, it is much easier and much safer to begin the dissection from the top and free the gallbladder from the liver and come down to the cystic duct, expose the cystic duct so that you can actually see it and then ligate off the cystic duct and cystic artery. That, you see, is what we are doing in this case.

I carry two drainage tubes down to the stump of the cystic duct, one about as large as a No. 30 F. catheter which is a thick rubber tube containing a wick of iodoform gauze. I allow this to project $\frac{2}{3}$ inch beyond the tube and come in contact with the stump of the cystic duct. The other is a rubber drainage tube about as large as a No. 10 F. catheter. I practically never close a cholecystectomy without drainage, even in the simplest case. I will ask you to follow me carefully as I close this abdominal wall.

My surgical nurse informs me that the pads and sponges are all accounted for. I proceed now to sew up, first, the peritoneum and the posterior sheath of the rectus with a running catgut suture, leaving at the upper angle simply enough space for the exit of the two drainage tubes. I then use four large tension button sutures (Fig. 84). These, as you see, consist of a large curved needle threaded with two pieces of silkworm-gut and at the end of these two pieces you see attached a pearl button about $\frac{7}{8}$ inch in diameter. The two silkworm-gut sutures are passed through two of the holes in the button and tied in a firm knot. The needle is passed about an inch from the incision through the skin and superficial fascia and anterior sheath of the rectus, first on one side and then coming out on the other, the needle passes first through the anterior sheath, through the superficial fascia and skin and comes out about an inch from the line of incision. I will now introduce four single silkworm-gut sutures, one above the first button suture. In passing this suture you will notice that I introduce the needle about $\frac{1}{4}$ inch from the line of incision. This passes in the same way through the superficial fascia, external sheath of the rectus. The second and third are passed in the same way between the button sutures, and the fourth, below the third button suture.

We now close with fairly fine catgut the external sheath of the rectus with a running stitch. In closing the wound and tying these stitches it is desirable to close the single silkworm-gut sutures first, then with interrupted sutures of black silk we close the integument and last, we tie the three button sutures. These should be tied fairly snugly, but not tight enough to produce

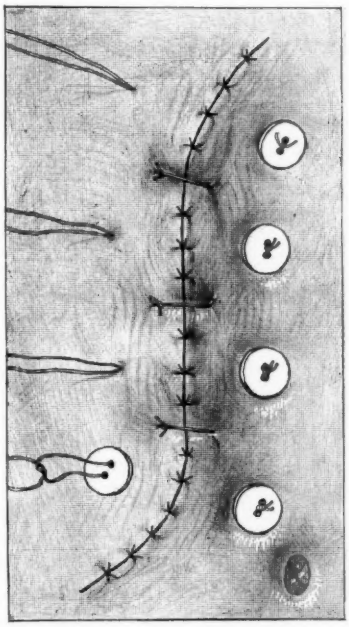

Fig. 84.—Closure of wound with button sutures.

a marked bulging of the tissue between the buttons. We have been using these button tension sutures in the clinic for about fifteen years in almost all of our abdominal incisions. They have proved to be of very great service. They have eliminated almost entirely the accident of evisceration following early removal of the stitches, an accident which is very apt to happen in operations on the stomach and operations on the bile tract,

especially in patients whose wound repair is slow and in patients who either have infections of the wound or have a postoperative lung complication, with resulting coughing, and of course, this may happen in individuals who vomit a great deal following operation.

To my mind the case of this patient is a very interesting one. He has been treated for peptic ulcer for years, when, in fact, his distress has been caused by gallstones and an infected gallbladder. I feel quite confident if he recovers from the operation, and I think he will, that the resulting history will show that he will have no further symptoms of stomach distress. We owe the correct diagnosis of this case not to any refined means of diagnosis, but to careful history taking and the gross clinical picture which suggested to my mind gallstones and gallbladder colic, and to the report of a simple flat plate which definitely showed gallstones. We have not employed the Graham test in this patient because I felt that it was unnecessary.

The second patient Mrs. Z. D., has been under the care of Dr. Ralph C. Brown. Dr. Brown made the clinical diagnosis of gallstone disease. She has been treated for a considerable period by other men for her stomach and for the distress which was regarded as being due to her colon. Dr. Brown believes that her abdominal attacks are gallstone colic, probably due to gallstones. The Graham test, when given intraorally, failed to show any filling of the gallbladder. This was checked with the intravenous test and that confirmed the fact that the gallbladder did not fill with bile and that is why it cast no shadow. Dr. Brown feels quite confident, however, that she has an obstruction of the cystic duct, that the bile does not pass into the gallbladder and that the evidence points strongly to a disease of the gallbladder as the cause of her acute abdominal attacks.

Dr. Herb is now anesthetizing the patient under the sequence of ethylene and ether. You see, I am making the same gallbladder incision and, as I open the peritoneal cavity, I find a greatly distended gallbladder. I bring the gallbladder into view

with a right-angle clamp attached to the fundus and separate the gallbladder from the liver. As I do this, I can feel the gallbladder and find that it contains a stone almost the size of a small egg. Very patiently and carefully I am separating the gallbladder so that I can bring it into view. It looks like a large pear attached to its stem, the stem being, of course, the cystic duct. I now change my position; you see, I have been operating on the right side of the patient. I find that in these cholecystectomies, after I have freed the gallbladder down to the cystic duct, I can complete the removal of the gallbladder much easier from the left side, so I now pass to the left side of the patient, and have my first assistant take my position on the right side. This gives a much better control of the situation; with a large fixed gallbladder like this it is very difficult to see into the depth of the peritoneal cavity from the right side, but from the left side and from a little below the umbilicus one can get an excellent view of the cystic duct and the common duct. I ligate the cystic duct with a small right-angle clamp and then put another small right-angle one about $\frac{1}{2}$ inch distal to this first clamp and divide between, removing the gallbladder containing the large stone. I ligate the cystic duct as in the previous case, introduce two drainage tubes and close as I did before.

My surgical nurse informs me that the pads and sponges are all accounted for.

I now split open this gallbladder, exposing this very large stone. As I split the gallbladder widely open several smaller stones come into view. The question may occur in your mind why this huge gallstone did not show in the *x*-ray plate. We have excellent *x*-ray plates in this case. Undoubtedly, no bile passed into the gallbladder from the use of the phthalein dye test and the gallbladder showed no shadow even after intravenous introduction of phthalein. The *x*-ray plates that we obtained were very good ones, quite as good as any flat plates in the previous case which showed the gallstones very clearly. The explanation is simple. This huge gallstone, as I cut it open, is nothing more than a great mass of cholesterine and

cholesterine shows little more shadow than bile itself, and sometimes less of a shadow than normal bile. In the other patient whom we operated on this morning the stone contained not only cholesterol, but it contained rather a thick layer of bile pigment, bilirubin, biliverdin, calcium, and some carbonate of calcium.

CONGENITAL PYLORIC STENOSIS

DR. BEVAN: The third patient on whom I shall operate this morning is a little patient of Dr. Parmelee. I shall ask Dr. Parmelee to make a statement as to the clinical picture.

DR. PARMELEE: *Infant J. Q.:* Four weeks old; fifth child; full-term infant that weighed 8 pounds, $14\frac{3}{4}$ ounces at birth. At two weeks weighed 10 pounds. Entirely breast fed. At three weeks of age began to vomit projectily. During first day vomited two or three times. Since then has vomited large amount after practically every feeding. Bowel movements which had previously been normal have become very infrequent and scanty and obtained only by use of suppository. During the last week he has lost over 1 pound in weight. Father, mother, and other four children are living and well. The second child had a very severe attack of pyloric spasm. He was under my care and treated in this hospital by stomach lavage and feedings by tube, and by the administration of atropine. He recovered and has been well ever since.

Upon examination we find an infant with large frame, but rather poorly nourished. The tissues show slightly reduced turgor. There is marked visible peristalsis over the epigastrium and on palpation a hard, firm tumor mass is felt in the region of the pylorus. The remainder of the physical examination reveals nothing abnormal.

Diagnosis of congenital hypertrophic pyloric stenosis was made on the basis of the persistent projectile vomiting, the loss of weight, the scanty and infrequent stools, visible peristaltic waves in the epigastrium, and finally, the palpation of a pyloric tumor. Operation was advised.

DR. BEVAN: I will ask Dr. Grulee, the head of the Pediatric Department, to present to you briefly the clinical picture of these cases.

DR. GRULEE: For many years now the pediatric department in this hospital has not advised operation for con-

genital pyloric stenosis where the diagnosis was not confirmed at the time of operation. This statement is made for two reasons: First, because we lay so much stress on the differentiation between pylorospasm and pyloric stenosis, the first being regarded as an entity to be treated entirely by nonsurgical procedures and the latter as a condition demanding immediate surgical interference. The second reason is that this statement supports our methods of diagnosis.

For practical purposes the only condition which enters very much into the differential diagnosis of congenital pyloric stenosis is pylorospasm so that principally one must consider the points of resemblance and the elements of difference between the two. In order to understand these we must have clearly before our minds the fact that both conditions are due to an overaction of the circular muscle coat at the pylorus, the only difference being that in pylorospasm this is not thickened, while in congenital pyloric stenosis it is several times its usual thickness. Therefore, the first point to be brought out is that in a given length of time, the picture of pyloric obstruction develops much more fully in congenital pyloric stenosis than in pylorospasm. It is, however, a strange fact which I cannot explain, that the severe symptoms, for example, projectile vomiting, frequently develop much earlier in pylorospasm (often in the first week of life) than they do in congenital pyloric stenosis. The progress in the latter is, however, much more rapid. A child with pylorospasm is much more likely to be irritable and peevish while the one with stenosis is rather frequently placid. Projectile vomiting and constipation are, of course, common to both, but likely to be more severe in stenosis.

For several years now, we have abandoned the use of the *x*-ray in diagnosis of this condition. Many years ago, we were able to show that this offered no help in distinguishing between severe cases of pylorospasm and cases of stenosis with mild symptoms. Our diagnosis now rests almost entirely upon the gastric peristaltic waves and palpation of the tumor. After a little experience, one can judge somewhat by the waves. In other words, in a given length of time these develop to a much

more marked degree in stenosis than in spasm. But after all, the final diagnosis must largely lie in the palpation of the tumor. This is felt well under the liver, deep down, at about the outer border of the right rectus muscle. It is felt best when the stomach is in contraction. In difficult cases it is our custom to force the child to take large quantities of a milk formula or of water. This is forced to the point where the child reacts with a forcible projectile vomiting. After the stomach has expelled its contents, the waves become much more evident and stronger and the pyloric musculature contracts down so that the tumor mass is, as a rule, easily palpated in the location named. It feels very much like an enlarged lymph gland.

One other point I should like to make with regard to these cases of severe vomiting. Whether this be due to the pylorospasm or pyloric stenosis, occasionally we encounter either before or after operation, a rather high fever. It frequently occurs when the thick cereal diet is employed as a treatment for severe vomiting in these babies. A rather interesting example of dehydration fever in connection with congenital pyloric stenosis is one which I met with some years ago. The child was seen in consultation, a diagnosis of pyloric stenosis was made and immediate operation advised. This was done by a competent surgeon at ten o'clock in the morning and was in every way technically successful. At eleven o'clock that night I was asked to see the child again because the temperature had risen to 106 F. After going over the child carefully, I could find no reason for the temperature nor could the surgeon suggest any. On inquiring as to what the child had been fed, I was told that it was given only normal salt solution all day. On demanding a taste of the salt solution, I found that the intern by mistake had ordered the stock salt solution rather than the diluted. The child was immediately given all the water it would take, with the result that in twenty-four hours the temperature was normal, and the child made an uneventful recovery. I have since seen one case where the temperature had risen to 107 F. This came down before operation, but rose immediately after to 107.8 F. The physician in this case, however, knew the

dangers of dehydration fever, and after an all-night battle, during which fluids were given to the child in every conceivable fashion, the temperature dropped and the child went on to complete recovery. Let me say in closing that I believe every case of congenital pyloric stenosis should be operated upon as soon as the diagnosis is made, and that the death rate in these cases depends more upon the time of diagnosis than upon any other single factor.

DR. BEVAN: You have heard Dr. Grulee's discussion of congenital pyloric stenosis and Dr. Parmelee's story of this little child, five weeks of age, who was normal at birth and then about two weeks ago began to vomit and has vomited every day since that time. It recently began to lose weight and has developed this picture which you see as the child lies before you, a marked peristalsis of the stomach. Dr. Parmelee has also been able to feel, in this case, a definite tumor. We shall, therefore, operate on this baby with the definite diagnosis of congenital pyloric stenosis and shall do a Rammstedt operation under local anesthesia. I have been doing this operation under local anesthesia for about fifteen years and it is very satisfactory. We have developed a technic which makes this Rammstedt operation one of the most finished pieces of surgery in the entire field of abdominal surgery.

You will notice that Dr. Herb, our anesthetist, has given the baby a little piece of gauze soaked in a sugar solution, covered with a rubber nipple, and that the baby sucks this as though it were a nipple of a nursing bottle. The baby is tied with bandages wound around its body and its extremities firmly to a padded board about 8 inches wide and about 3 feet in length. This controls any movement of the baby completely and does so without injury to the little patient. You will notice that I introduce the needle in the upper angle of the proposed incision and the child makes a little cry. Now you see that this is momentary and it is sucking its little gauze pad with sugar solution quite contentedly. I now infiltrate the skin over the right rectus muscle from the costal arch down to the umbilicus with our usual $\frac{1}{2}$ per cent novocaine solution in distilled

water to which we have added adrenalin solution, making it a 1 : 200,000 solution. The tissues are now quite anesthetized from the costal arch down to the umbilicus at the junction of the inner third and outer two thirds of the rectus muscle. I divide the skin and superficial fascia and external sheath of the rectus (Fig. 85, A). I now infiltrate the rectus muscle and split the rectus in the same plane as the external incision. I very carefully, with a very fine needle, infiltrate the posterior sheath of the rectus and the peritoneum. I now divide the posterior sheath and the peritoneum for the upper two thirds of this

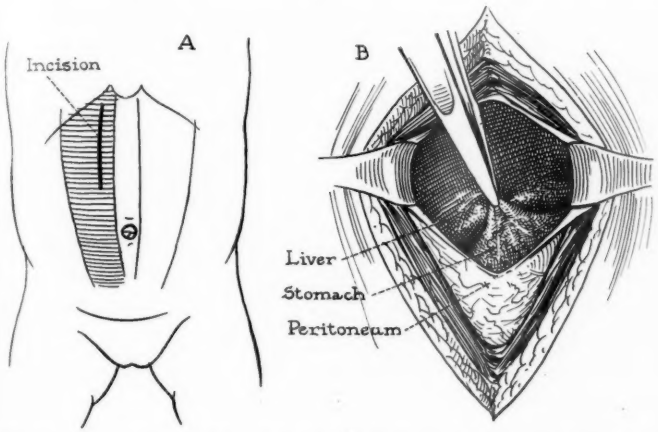

Fig. 85.—Operation: A. Line of incision. B. Exposure of stomach and liver.

exposure's extent in this incision and simply divide that part of the peritoneum and posterior sheath of the rectus muscle that covers over the liver (Fig. 85, B). This is one of the most important steps in the operation. The liver in a child extends down lower than it does in the adult, extending just below the costal arch. You see, as I now open the peritoneal cavity, there is nothing in view but the liver. No intestines are exposed, no omentum is exposed. This technic will prevent evisceration of the child, which is bound to occur unless this technic is followed. I now produce a smooth pair of dissecting forceps with-

out any teeth and push up the end of the liver, exposing the pyloric end of the stomach. I grasp the pyloric end of the stomach with these smooth forceps and draw it into view. As I draw this into view out of the incision I also bring out a tumor about the size of a small cranberry involving the pylorus. I grasp this tumor between my thumb and finger of the left hand and split the peritoneum and the superficial layer of the musculature of the tumor with a knife for a distance of about ½ inch (Fig. 86, C). I then take a pair of small mosquito artery forceps and introduce them close into the line of this incision,

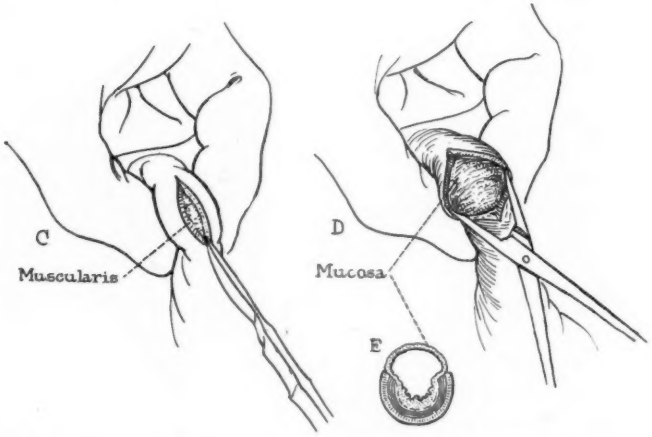

Fig. 86.—Method of exposure and spreading of hypertrophied musculature which forms the tumor.

separate them and spread apart the hypertrophied musculature which forms the tumor (Fig. 86, D). In doing this, you will gradually see come into view at the top of the tube mucous membrane and submucosa which is found at the junction of the stomach and duodenum beneath the pyloric tumor. You will notice that I am very careful not to spread apart the very lower part of the tumor toward the duodenum because I have found if I attempt to separate the tumor too widely, I am liable to tear an opening into the duodenum through the mucous membrane. There is very little danger of this occurring in

separating the tumor toward the stomach side. You will see that this tube comes very freely into view. I examine it. I examine the line of my incision and find no bleeding that requires any ligature. I, therefore, drop the little pyloric tumor which has now been divided as I am describing it to you back into the peritoneal cavity and close both the peritoneum and the posterior sheath of the rectus with a catgut suture, and then put in two tension button sutures and three single silkworm-gut sutures, one above and one below the buttons and one between the two button sutures. I then close the anterior sheath of the rectus with catgut and the skin with fine silk.

I want to call your attention to the dressing which I am applying in this case. I am using a dressing of Lassar's zinc oxide paste. This is to prevent soiling the wound with urine and feces and is much the best method of dressing an abdominal wound in an infant in the diaper age. Over this dressing I am putting some gauze and then this adhesive, 3 inches in width, which surrounds the entire child. I regard this as very important because it prevents any undue tension on the stitches. One of the risks of an operation of a case of this kind is owing to the fact that the child has been starved for a considerable period and wound repair is slow, so that it will be necessary to maintain good approximation for a considerable period before we remove the stitches, usually twelve to fifteen days. If you do not do this evisceration may take place when we remove the stitches.

I particularly want to emphasize the importance of this technic that I have employed in this case in handling these little patients. I am glad to say that our pediatricians are in perfect harmony with the surgeons in the matter of giving to these little half-starved patients the benefit of the surgical relief. They are all converted to the position that when a true case of pyloric stenosis develops it should be handled by a surgical operation. Our mortality has been exceedingly low since we have adopted the Rammstedt method of operation and since we have introduced the employment of local anesthesia. These patients run a much less risk from surgical operation than they do from the ordinary management aimed at the relief of this condition by special diet.

APPENDICITIS

THE next case that we shall do this morning is an intricate case of appendicitis. The patient is one of my interns and the story is very much like the shoemaker's wife and his children, who go without shoes. This young man was working in the surgical service of the hospital and for a couple of days had a good deal of distress in his abdomen, but he was very strong and had been very well. He hated to give up and had no one examine him. I came to the hospital one morning and found him in great pain with a rigid abdomen, more on the right side than on the left. The leukocyte count was 15,000, the temperature 101 F., and the pulse about 100. The urine was normal. I immediately sent him to the operating room and did the usual muscle-splitting incision for appendicitis. I found a large appendix which had ruptured and was pretty well walled off in the groove between the parietal wall and the cecum. When I lifted out the appendix there was an abscess containing about ½ ounce or an ounce of pus which discharged its contents on the gauze pack. The cecum at the point of attachment to the appendix was indurated and there was a good deal of fibrin and lymph surrounding the entire focus of infection. The appendix was removed in the usual way and ample drainage provided. The boy went on, apparently, to recovery. He left the hospital, however, still discharging through the sinus at the site of the drainage tube. This finally healed up and he was apparently all right for about six weeks. Now he comes back to me with the statement that he has had some pain in about the center of the scar and a good deal of tenderness. There is evidently an induration, probably an abscess fairly deeply situated about the center of the line of incision.

Under ethylene I shall make an incision through the center of the scar. As I do this you will see that the tissues are very edematous. I am going through edematous, soft tissue for a

distance of about 1½ or possibly 2 inches. Now I come down to the abscess containing probably a dram or two of pus. I am outside of the general peritoneal cavity and I do not intend to open into the peritoneum, but simply drain this pus pocket. I introduce a rubber tube containing iodoform gauze and pass a strip of iodoform gauze down into the abscess and shall use moist dressings. We know that the appendix has been entirely removed.

I can see no definite explanation for the occurrence of this abscess in the scar. No nonabsorbable suture was employed. We apparently did not leave behind anything in the way of a gauze pack or a sponge. It is possible, of course, that a fecal stone may have escaped from the perforation and may have remained undiscovered in the tissues at the time of operation. Another possibility, of course, is that of a tuberculous complication. I think that is hardly probable because of the sudden onset and acute character of the symptoms in this first attack. On any account, I would emphasize that the wise thing is not to go any further than opening the abscess at this time.

After History.—An interesting sequela to this case is that after operation hot dressings were applied and the gauze and drainage tubes gradually removed. At the end of about six or seven days the patient noticed something protruding from the fistulous tract left after the removal of the drainage tube and with a little pressure he pressed out from the tract a fecal stone about the size of the end of my little finger. It was not very hard, but rather putty-like in consistency. This undoubtedly was the cause of the recurrence of the abscess. Since that time I have taken a small gallstone scoop and very gently scooped out this tract. I thought there might be another concretion but none developed and he went on to, apparently, a complete recovery, after this opening of the secondary abscess.

DOUBLE UNDESCENDED TESTES

THE first case I shall operate upon this morning is a boy, nine years of age, with double undescended testes. The boy you see is a well-built, sturdy youngster, well developed, apparently, except that there are no testes in the scrotum. These

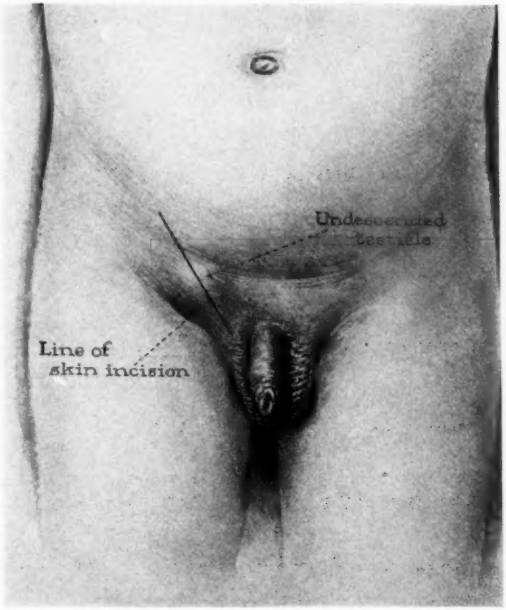

Fig. 87.—The skin incision.

can be readily palpated just at the external ring and in the end of the canal. I shall do the operation for undescended testes which we have developed here in this clinic. I did this operation first, back in 1898.

The patient is now anesthetized with ethylene and I shall outline to you the details of the operation.

scrotum without any tension whatever, looking very much the same as on the other side (Fig. 92).

You will see that we have succeeded in bringing the testicle down into the large pocket which we have made in the right side of the scrotum, so there is no tension whatever on the cord, no tendency for the testicle to return into its abnormal position in the groin. The question arises as to whether we

Fig. 92.—The result of the operation.

shall operate on the other side this morning. You will notice that the scrotum was very rudimentary and that the testis that I have put back into it is of normal size for a child of this age. I think it would be a mistake to operate on the left side at this sitting, because I have found by experience that there is some risk in overdistending the scrotum, the risk of edema and a good deal of swelling and the result in the cases where this occurs is not as good as in cases where we operate on one side at a time.

I am, therefore, going to be content with replacing the right testis this morning and along about Christmas holidays I shall operate upon the other side.

The second case is a young man of nineteen. This patient I have already operated two months ago on the right side and I shall do the second or left side this morning. You will notice that this patient is a well-developed young man. The external genitalia are normal. The testis which I brought down two months ago is freely movable in the right side of the scrotum without tenderness and is of fairly normal size. I shall review briefly as I go on with this work, the important steps of the operation.

The incision, as you see, is the regular one for the ordinary operation for the radical cure for hernia. We divide in the same way the skin, the superficial fascia, the intercolumnar fascia, the cremasteric fascia, and the infundibular fascia. We come down to a large peritoneal sac which leads well down toward the scrotum and well up into the groins. In this sac is a fairly well-formed testis. I cut through the peritoneal sac from the surrounding tissues, splitting the external oblique up to the internal ring, and then divide the peritoneal process about 1 inch below the external ring at right angles to the cord. I blow up the tissues of the cord with a hypodermic needle and a syringe containing normal salt solution, drawing the peritoneum away from the cord to make a division of the peritoneum easier. I have completely divided the peritoneum, and with a dissecting forceps stripped the peritoneum above the transverse incision up to the internal ring. I ligate the vaginal process as high up as the internal ring. I then split the peritoneum from the line of incision into the vaginal process from the cord, being careful not to injure the vas or blood supplies. As I do this, you will notice that I lengthen the cord about 4 inches. It is not quite as long as I desire. On spreading the cord out over my gloved finger, I find here a dense connective band which I tear across. Here is another, and I tear this connective band tissue across. This enables me to obtain probably another $\frac{2}{3}$ inch in length. I then close the vaginal process with a purse-string suture just above the testis,

making a tunica vaginalis. Then with the fingers I make a pocket in that side of the scrotum and pack that side of the scrotum with a good-sized piece of gauze, wet in normal salt solution. By placing this gauze into the scrotal pocket on the side I make a large cavity, much larger than the testis, so I can now drop the testicle into it without any tension. The testicle is now in its normal position in the scrotum and I prevent its returning into the groin by a purse-string at the entrance of the scrotum, leaving plenty of room, of course, for the cord. It is unnecessary for me to emphasize the fact that the cord is not included in the purse-string suture but is situated well behind the purse-string suture. We now return structures in and about the canal, leaving the cord deeply situated in the canal. I close over the cord first with the conjoined tendon to the shelf of Poupart's, then the external oblique to the edge of Poupart's and finally with a very fine catgut suture sew together the edges of the deep layer and superficial fascia and, last, the skin incision is closed with interrupted black silk.

Before leaving the subject this morning I want to show you another case. This is a patient upon whom we operated some months ago. You see, this is a very fat boy. He is, however, a brilliant boy. He is not at all backward in his classes. His external genitalia are small and the testes are now in their normal position. I have placed him on small amounts of iodine and he is taking Lugol's solution twice daily. Under this he is losing a little weight and I believe he is developing into a more normal condition.

CARCINOMA OF THE BREAST

THIS patient, a woman of forty, came to the clinic with a tumor in her left breast. The tumor is irregular, about $\frac{1}{2}$ inch in diameter and possibly $\frac{1}{3}$ inch in thickness. It evidently has no definite capsule. I can move it in the breast tissue and yet there is a certain amount of fixation to the mammary gland tissue. I am uncertain in my own mind as to the diagnosis. My first impression was, because of a fair degree of mobility, that it was probably a localized area of chronic, cystic mastitis or Schimmelbusch's tumor. A second impression is that the diagnosis is doubtful and that it is difficult to rule out carcinoma of the breast. It is very important for us to know the character of this tumor before we proceed with the operation.

The patient is now anesthetized under ethylene. We do practically all of our operations on the mammary glands under ethylene. It acts admirably in this type of case. I think one may say that it is an ideal anesthetic for operations on the breast. I shall cut down directly on this tumor mass, dissect it out rather widely, look at it on gross section, and obtain a rapid frozen section. I now dissect out the mass widely, as you see. There is some bleeding which I control with forceps and ligatures. While I examine the breast I shall have one of my assistants close the wound which I have made very tightly, so that there will be no oozing and we will have the field of operation sterilized before we continue whatever procedure we decide upon.

As I examine this piece of tissue and cut across it, it gives me the impression that it is probably a small carcinoma. Dr. Apfelbach, who is here and who is in charge of our pathologic laboratory, will take this piece of tissue and make a rapid frozen section and bring in a report within a short time. In the meantime, I want to say that I have found in our clinic that we can make in probably 90 per cent of the cases a clinical diagnosis

as to whether these breast tumors are benign or malignant, and proceed on that diagnosis to do a radical amputation or a local excision of the tumor, depending upon the character of the growth. There is, possibly, a group of, say, 10 per cent of these cases in which the diagnosis is not sufficiently clear to decide as to the character of the operation without making a biopsy and examining the gross specimen and obtaining, as we very frequently do, if it seems necessary, a frozen section for microscopic examination. As a matter of fact, the gross appearance of the tumor is in a very large majority of the cases, perfectly definite and determining so that we can decide quite definitely whether we have a benign or a malignant growth to deal with. I do not place a great deal of importance, however, in the ordinary case on making a frozen section. We rely, certainly in more than 95 per cent of the cases, on the gross pathologic appearance. It is worthwhile, however, especially in doubtful cases, to obtain a good frozen section to check up the diagnosis.

Dr. Apfelbach has now returned with his report and states that the growth is definitely a scirrhous carcinoma. We shall, therefore, proceed at once with a radical breast amputation. I would like to present to you first the general principles upon which this operation is planned. It is not wise to adopt any set method of incision for these radical breast amputations because the incision should be so planned that the block of tissue removed will have, as nearly as possible, the primary lesion in its center. Often the tumor is at the periphery of the breast and in such a position that the ordinary Halsted incision or the Kocher incision would cut across the tumor if the incision were made in the classical way. Therefore, we adopt the simple plan of making the incision in such a way that the primary lesion is in the center of the block of tissue and at the same time we try (Fig. 93), in almost all the cases, to save enough skin to make a primary closure. We seldom today close, as we did years ago, these radical breasts with huge skin-grafting operations. In this case the tumor is a little to the outer side. We save more integument on the inner side near the middle and plan the incision in the overlying skin fairly widely, and with the

primary lesion about the center of the area removed. We shall remove the entire breast and as I proceed you will see that I separate the integument from the mammary gland and am very careful not to leave any of the mammary gland tissue, under-

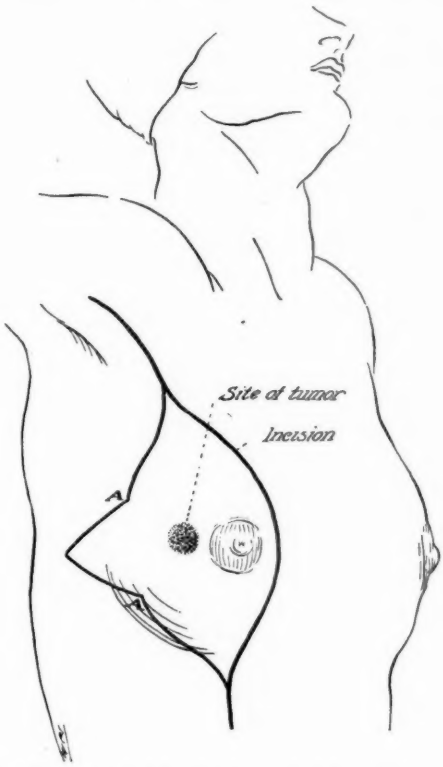

Fig. 93.—Line incision for removal of breast tumor.

cutting the skin as we proceed. I carry the incision from about the origin of the pectoralis major into the humerus and down to the inner side of the nipple over the entire breast and down over the origin of the rectus abdominis muscle. As I dissect off the skin and a thin layer of superficial fascia I now expose

the pectoralis major, especially that part of it that arises from the sternum and from the costal cartilage. The incision on the outer side of the breast is carried out in the same way, except that as we dissect off the integument and superficial fascia, we come down to the digitations of the serratus magnus muscle, and expose the outer edge of the pectoralis major. I now lift up the pectoralis major and dissect it from its origin at the costal cartilages and from the sternum. You will notice that as I do this I clamp the muscle close to the costal cartilages and the ribs with strong artery clamps as I proceed with the dissection. I do this in order to control the bleeding from the intercostal branches of the internal mammary arteries. I am now able to elevate the breast and the pectoralis major muscle off the thorax, exposing the pectoralis minor. I leave, as you notice, a portion of the pectoralis major that takes its origin from the clavicle. As I make this separation between the clavicular and sternal origins of the pectoralis major I have to clamp some good-sized vessels and nerves that come out from the costocoracoid membrane and which supply the pectoralis major and mammary gland. It is now necessary to divide the insertion of the pectoralis major into the outer lip of the bicipital groove. I do this with a knife and I find it necessary to clamp several arterial branches in this muscle mass in order to control the bleeding. It will probably not be necessary to make any ligation of the vessels. There remains the dissection of the axilla. It is very important for us to have an easily recognized guide for this dissection and that guide is the axillary vein. I am now dividing all the fascia over the axillary vein. You see this large blue vein comes into view. With retractors holding the pectoralis minor and pectoralis major out of the way so as to expose the axillary vein, I now make a dissection and remove all the axillary fat and the lymphatics, being very careful not to injure any of the important nerves. It is, however, necessary for me to divide the two costohumeral nerves which run across the axillary space. These, however, are not important because they are simply cutaneous nerves. You see, however, that it is necessary to clamp and ligate a number of branches of both the axillary vein and axil-

lary artery. The contents of the axilla are removed with the large block of tissue comprising the overlying skin, the mammary gland, the pectoralis major muscle. You will see exposed in the axillary space the axillary artery, the brachial plexus of nerves, and the axillary vein. I want to note that it is not wise to pick off from these structures every little vestige of connective tissue and fat and leave these structures exposed in a very bare way. That I do not regard as good surgery. I think if one carries the dissection to too great an extreme he is much more apt to get an interference with return lymphatic and venous circulation with the resulting edema of the arm. I want a good, clean-cut dissection, but not one carried to the extreme. There remains now control of all the bleeding points with ligatures wherever it is necessary. As a rule, however, I use very firm artery clamps and by the time the operation is completed very few of the vessels need ligating. You will notice that I removed the clamps and that there is no bleeding after their removal. If there is any bleeding the clamp is reapplied and that particular point ligated, so probably three fourths of the vessels clamped do not require any ligation. We now close the skin incision with interrupted silkworm-gut and place them about 2 inches apart. Before applying my sutures, however, I make this little stab wound just beneath the axilla and introduce a soft, collapsible tube for drainage of the axillary space. Finally, a very copious, sterile gauze dressing is applied very firmly with a sterile gauze roller and over this a starched bandage to retain the dressing in place. The patient, from the very first, has the use of her arm below the elbow. We rather insist upon this. In spite of the huge extent of the wound in these breast amputations, there is very seldom any shock after the amputation, even though the patient has lost a good deal of blood. Patients stand the loss of blood in these amputations very much better than they do a loss of blood in a laparotomy. I cannot tell just why, but I have noticed this, and a great many other surgeons have been impressed by it.

The mortality in our breast amputations has been but a fraction of 1 per cent. We have lost 3 cases in certainly more

than 500 cases of radical breasts. One of these was from secondary hemorrhage in a patient upon whom I did a palliative operation for a huge, ulcerating mass. The patient was a Christian Scientist who had allowed the tumor to develop until there was a very foul-smelling, ulcerating mass, and she finally determined to have it removed surgically, if possible. I did the operation with some hesitation, but felt that I probably would be doing the patient a service, though I recognized from the standpoint of permanent cure that the case was definitely inoperable. On about the tenth day a sudden giving away of the axillary vessels occurred and a secondary hemorrhage followed which terminated fatally. The second case was one upon whom I had done a very extensive breast amputation and left a large area for skin grafting. The patient made a good recovery from the amputation, but she died from erysipelas secondary to the skin grafting operation done to close the defect. The third was a patient with a very bad heart and with a not very extensive carcinoma who died on the fourth or fifth day from a heart attack. No postmortem was obtained, but the clinical picture was that of pulmonary embolism.

Usually the patients are sitting up on the day following operation, out of bed for the greater part of the day. The stitches are usually removed on the eighth or tenth day, and the patients, as a rule, will leave the hospital by the twelfth to the fourteenth day. I am very firm in my opinion that proper *x*-ray treatment after these operations is of very great value, and adds considerably to protection against recurrence. There is, to be sure, danger in the after-treatment with the *x*-ray. We have seen three patients die from massive *x*-ray doses which produced a fibrosis of the lungs, fortunately, none of them in our own hospital or in our own service. In these cases we obtained postmortems and found a very extensive fibrosis with fluid in the pleural cavity, and in two of them we did not find, even by very careful examination, any carcinoma cells in any of the tissues of the body. These experiences have convinced us of the great risk of massive doses in the after-treatment of carcinoma of the breast. The dosage should be

moderate and carry no such risk of lung injury or risk of burning the skin. We unfortunately have seen a number of very severe burns following the use of *x*-ray in carcinoma of the breast. With care, I am satisfied that the *x*-ray offers a good deal of additional assurance against the recurrence of the carcinoma. Our ordinary routine is to give the patient about eighteen treatments a week apart and sometimes a vacation of a month in the middle of the series. The logic of this procedure is perfectly plain. We have repeatedly seen nodules the size of my little finger or the end of my index finger which have developed in the scars of breast amputations disappear under moderate *x*-ray treatment and it is fair to suppose that if these nodules of carcinoma melt down under *x*-ray and disappear, the microscopic group of cells from which these nodules spring would be much easier influenced by the early application of the *x*-ray.

What is the prognosis of carcinoma of the breast today? The woman who comes to us with a carcinoma of the breast where the lesion is limited to the block of tissue which can be removed at operation and where no evidence, gross or microscopic, of axillary lymphatics is found, the prognosis is 75 or 80 per cent of permanent cure. This is in no way an exaggeration. Where, however, at operation the axillary glands are found involved, the percentage of permanent cure immediately drops down to less than 10 per cent. Unfortunately, the majority of our cases come to us at a time when the axillary glands are already involved. As a result we are probably curing today, the country over, in all the best clinics, possibly 30 or 35 per cent of these carcinoma cases. This fact speaks very strongly for the propaganda that is being carried on by the Society for the Control of Cancer which will educate the public and the medical profession to recognize the fact that carcinoma is curable and that carcinoma of the breast, particularly, is curable if the patients are brought to us at a time when the lesion is still limited to the breast.

I want to show a patient upon whom we operated six months ago. This big, strapping fellow, 6 feet 2 inches or more, was injured during the war. He was a marine. He was in very

active service in France, had his leg shot off, and he is wearing an artificial limb. He has good courage and he is employed in a bank. He returned to his job, married, and has four children. He was very happy and contented until about eight months ago when he began to have pain in the upper part of his left chest and back under the scapula. He went to the Naval Hospital in Washington and there they found a sarcoma of the fourth rib,

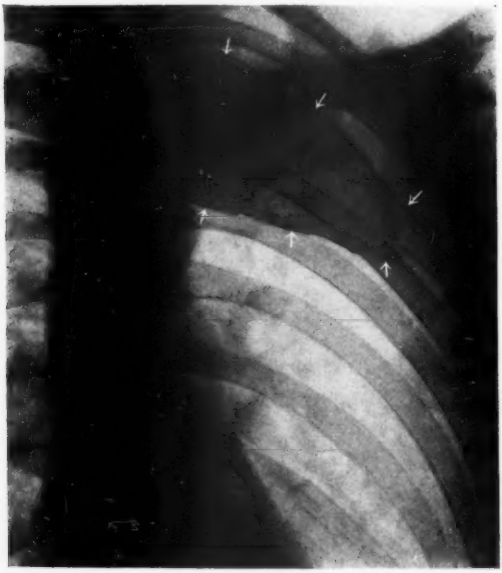

Fig. 94.—Roentgenogram showing sarcoma of the fourth rib, about 4 inches in length and about 2½ inches in width.

about 4 inches in length and about 2½ inches in width, as shown in the *x*-ray (Fig. 94). They were rather loathe to operate upon him and gave him the name of four different surgeons to whom they would refer him. Living in Chicago, he, naturally, would come to me as I was one of the four mentioned. The case looked like a very difficult problem. I decided to operate upon him. It was necessary to expose the rib posteriorly and it was

necessary to make this huge incision 15 inches in length just to the inner border of the left scapula, extending up to the neck and well down the side of the chest. I had to divide the skin and superficial fascia, the trapezius muscle and the serratus major and serratus minor and draw the scapula away from the chest to expose the fourth rib. I then split the periosteum over the fourth rib the entire length of the tumor, 4 or 5 inches in length and very close to the head of the rib. I then found that I could readily cut down through the thin shell on to the tumor. The tumor bled terrifically. I rapidly cureted it out with a large curet and stuffed it with iodoform gauze. It was impossible to remove the tumor without going into the chest cavity and involving the pleura and the lungs and this I did not want to do. After cureting out soft structures and tissues and leaving simply the shell of the bone between the tumor and the lungs, I packed it with iodoform gauze and closed the incision except for about 4 inches through which the gauze and drainage tubes passed. I began immediately *x*-ray treatments. I have had considerable experience with these vascular sarcomas of the bone. Some of them are very malignant, others are less malignant and more amenable to treatment. He has had about ten *x*-ray treatments and he has regained his old weight, his old strength, and the *x*-ray shows that there is a new deposit of bone in the position occupied formerly by the old tumor. He is still continuing with the *x*-ray treatments. I am quite enthusiastic about the outcome of the case so far and I am very hopeful that the result may be permanent. My colleague, Dr. Dallas B. Phemister, at the Billings Hospital, University of Chicago, has had a number of similar experiences of the disappearance of these vascular bone tumors under proper *x*-ray management.

CLINIC OF DR. KELLOGG SPEED

PRESBYTERIAN HOSPITAL

TUMOR OF THE CHEST WALL

FOR the registry of thoracic tumors I have just sent in a report of this patient whom I show you today apparently in good health. True tumors of the thoracic wall are rare; their careful study and follow-up, their compilation by the committee on thoracic tumors will eventually lead to instructive information on their pathology and treatment. This patient has been thoroughly studied, and I offer you a copy of my report to the committee:

History.—She is white, Polish, thirty-five years of age, a housewife.

Chief Complaint.—Tumor mass in the right chest wall which is painful and interferes with sleeping and work.

Past History.—Many years ago a small tumor mass had been removed from her left breast. This breast had never given any trouble whatsoever. No history of respiratory infection was obtained. About twelve years ago the patient had jumped from a street car with a resulting injury to the right side of the chest; she also received a skull fracture. She does not know whether ribs were broken or not. There had been no history of cough, loss of weight, or ill health.

Family History.—At the time of admission she had been married two years; had one live child ten months old and history of one miscarriage in the fifth month. Family history negative as to neoplasms.

Present Illness.—Seven years before coming to the Presbyterian Hospital a tumor mass began to grow on the right side of chest wall on the anterolateral aspect of the lower ribs. Within five years the tumor had reached the size of a baby's

head. This was removed surgically, apparently by a right rectus abdominal incision as that is the only scar present on the chest or abdomen (Figs. 95, 96). There is no way of obtaining the hospital record of this operation or of the pathology found at that time.

Two and a half years after this operation she came to the Presbyterian Hospital seeking admission on account of the present illness which consisted in a recurrence on the right side of the tumor mass the size of a large grapefruit. She had pain in the chest wall and along the incision of the previous operation in the right rectus muscle. This pain was not constant; not necessarily worse at night but was aggravated by lying on the affected side, disturbing her sleep very much. There was no cough; the patient was overweight.

Physical Examination.—April 24, 1924.

(*a*) General physical examination revealed a normal slightly obese woman with no evidence of tumor in either breast. There was a tumor on the right side of the chest wall the size of a grapefruit, firmly attached to the lower right ribs anteriorly. This was rounded, solid, had the consistency of cartilage and extended over the area from the sixth to the tenth right ribs inclusively. The scar of the previous operation was median to the tumor in the right rectus region, 7 inches long, extending directly downward from the costal margin.

(*b*) Temperature on admission was 100 F.; hemoglobin 90 per cent; white blood cells 7500; Wassermann negative; blood pressure 130/80; urine normal. *x*-Ray on April 25, 1924 showed no apparent pathologic changes in the ribs or other bone fields. Left lung field as a whole was more dense than the right, probably due to an old pleurisy; no evidence of metastases in the lungs.

Diagnosis.—A diagnosis was made of a probable malignant tumor; sarcoma of the right chest wall. No biopsy was performed but the patient was prepared for operation.

Treatment.—An operation was performed under ethylene on April 28, 1924. A transverse incision 10 or 12 inches long was made over the right costal margin from midline lateral and

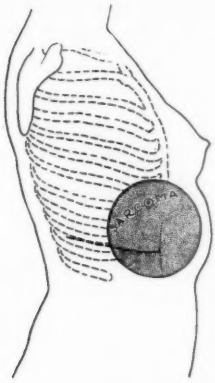

Fig. 95.—Schematic drawing representing the position of the tumor mass on the lateral anterothoracic wall. The dotted line represents the scar of a previous laparotomy. The interrupted line represents the incision made for the removal of the tumor mass and its attached ribs.

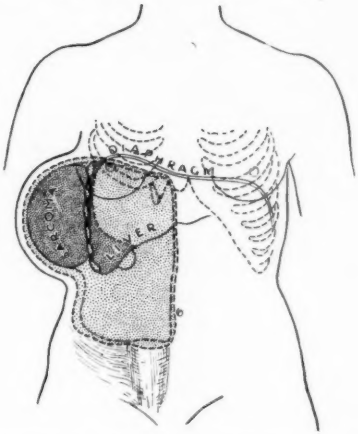

Fig. 96.—Schematic drawing representing the anatomic position of the tumor mass showing its infiltration around the sixth to tenth ribs which are represented as cut away. It has also been attempted to show the involvement of the liver representing approximately the amount of this gland which was removed with the tumor. The stippled areas on the abdominal wall represent the amount of muscular and fascial tissues removed from the front of the abdomen.

backward. The area of the skin immediately over the tumor was left attached to it (Fig. 97). The skin was dissected upward as far as the sixth rib and downward to the crest of the ilium. The tumor mass, which seemed to originate from the lower ribs and costal cartilages was dissected free with the attached abdominal muscles on the right side. This necessitated removal of all muscular layers including part of the rectus abdominis down

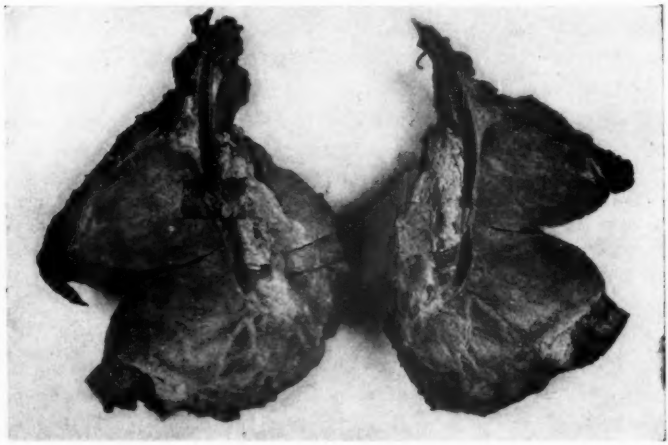

Fig. 97.—Tumor of the thoracic wall split open showing the cut through rib which lay in its center. The extent of the tumor around the rib and into the liver substance which lies toward the center. and the thoracic wall which lies on the outer side is quite evident. At the bottom part of the figure is seen some of the attached diaphragm wall. The mass of the tumor looks like fibrous tissue.

to the iliac crest and laterally to the posterior axillary line. Above, 5 ribs were resected wide of the tumor mass. The dissection was carried forward to include most of the costal cartilages and approximated the lower margin of the pericardial sac. The right pleural cavity and the peritoneal cavity were both widely opened. No adhesions to the parietal pleura or diaphragm were found about the right lung. The right lung retracted, was seen to be occupying about one-half of the right

pleural cavity expanding and contracting with moist, glistening pleural covering.

To remove the tumor mass the anterior and lateral insertion of the diaphragm had to be cut away from its lateral and rib insertion for at least 10 inches. In the abdominal cavity it was necessary to resect through adherent omentum and the gastrohepatic ligament (Figs. 98–100). The tumor was found densely adherent to and infiltrating the anterior surface of the

Fig. 98.—Another view of the tumor mass showing its main portion cut through bulging out from the subcutaneous tissues covering it. At the bottom of the photograph is seen liver tissue. Above that are layers of pleura and diaphragm involved in the growth of the tumor mass.

right lobe of the liver. This portion of the liver was resected with the electric cautery and mattress catgut stitches were inserted in the cut liver surface to control bleeding and a narrow gauze pack was inserted down to the liver. The free, retracted, cut edge of the right diaphragm was then sutured to the remnants of the transversalis fascia and the sheath of the rectus, by pulling the diaphragm well down over the dome and anterior surface of the liver. To close the pleural cavity the subcutaneous tissue of the chest skin flap was next sutured to the diaphragm. The skin

Fig. 99.—*x*-Ray film of the specimen of thoracic tumor after its removal. This shows the portion of the five ribs removed and the density of the tumor as a whole. There is very little evidence that the bony part of the rib has been directly involved and from the film alone one would be inclined to diagnose a periosteal fibrosarcoma.

Fig. 100.—Lateral view of the removed specimen of thoracic tumor showing pieces of five ribs and tumor, with liver substance attached. The specimen has been slit open at the cut surface and used for microscopical section. The tumor density is not as great as the ribs, but is considerably more than the average soft parts.

was then closed over the abdominal cavity with no peritoneum or muscular tissue remaining beneath. A tubular drain was inserted laterally. A microscopic diagnosis of fibrosarcoma was made.

x-Ray Treatments.—None were ever given.

Gross Features of the Preserved Specimen.—The growth is deeply placed in the front wall of the trunk close to the sternal portions of the sixth to the tenth right ribs inclusive. About 10 to 15 cm. of these ribs removed with the tumor lie behind it. The long axis of the growth, 12 cm., is parallel to these ribs. The other dimensions at right angles to the long axis are 8.5 and 10 cm., this last the distance front to back. Toward the vertical midline of the trunk, the tumor extends to about opposite the sternal ends of the costal cartilages of these ribs.

Below the costal caudad border of this part of the right thorax wall and toward the midline of the trunk, there is very little of the deeper layers of the abdominal wall dorsal to the tumor. Here the growth has pushed the ventral adjacent peritoneum up under the margin of the costal arch against the diaphragm and liver so that all are bound tightly together, the tumor in front, then the diaphragm and finally the liver. Thus it has come about that with removal of the tumor a layer 13 mm. where it is thickest and 5 cm. in the other dimensions has also been removed from the front of the liver close to the furrow between the right and left lobes.

Histology.—The tumor is a slowly growing recurrent fibroma, of the type sometimes called "desmoid"; sometimes called fibrosarcoma. They often weigh several pounds and grow to be large tumors in the broad ligaments as well as in the ventral wall of the trunk. Occasionally they are encountered in the neck. In the dorsal part of the trunk they are exceedingly rare. The inguinal region is their usual location, In microscopic preparations from many places only fibroblasts, fibers, veins with apparently no walls and arterioles with no muscular fibers in layers are met with. Collogen is very abundant. There is no invasion of adjacent structures by the tumor. They are simply pushed aside or compressed by expansile growth.

Progress of Case.—4/30/24: Gauze pack removed; no bleeding; condition fairly good; resonance present over right lung.

5/5/24: Wound discharging small amount of purulent material; skin reddened and a little brownish but looks as if it would hold; alcohol dressings. The patient was quite dehydrated in spite of efforts at proctoclysis.

5/6/24: Some pain in the right side during respiration; chest still resonant, abdomen soft and no signs of peritonitis; two days later tubular breathing was heard on the right side.

5/12/24: All stitches removed; wound granulating a little; some infectious discharge.

6/14/24: Left hospital, wound practically closed; patient walking, wearing a supporting abdominal bandage. She ran postoperative course of temperature up as high as 100 F. *x*-Ray showed no pneumothorax. Breasts normal; no pleural effusion was demonstrated.

Date of last examination was December 7, 1928. Her condition at that time gave no evidence of recurrence of the tumor mass on physical or *x*-ray examination. The right diaphragm moved freely and normally; was raised outward and upward toward the chest wall. Patient still had a large postoperative hernia involving the whole right side of abdomen, retained quite satisfactorily by the abdominal support she wore.

In addition to the fact that this patient has gone six years since the removal of a malignant sarcoma, the result enters into a discussion of the physiology of the action of the diaphragm, the intercostal and the abdominal muscles, and postoperative pulmonary complications. According to Lemon, quoting many previous authors, the development of the diaphragm is complex. It originates in the septum transversum near the head of the embryo and migrates caudally. Broman gives other sources of origin of the diaphragm which include the mesentery, pleuroperitoneal membrane and the lateral body walls.

If the diaphragm originates partly from the lateral body walls anatomically one would expect that it would have two sources of motor nerve supply, one from the phrenic or fifth cervical segment, which comes to it from the septum trans-

versum, and the other from the source supplying the muscular body wall which contributes to its formation. Kingsley in the comparative anatomy of vertebrates, believed that the ventral portion of the diaphragm took origin from the rectus abdominis muscle.

In the change from pronograde to orthograde types of vertebrates the diaphragm certainly took on two functions: first came the effort to increase intracelomic pressure to aid the abdominal muscles in overcoming the sphincters, and second, the automatic respiratory function. In man the diaphragm acting with other muscles enlarges the long diameter of the chest cavity. It moves downward during inspiration pressing on the abdominal viscera distending the abdomen, and raising the margin of the ribs outward and upward. According to Cunningham, "The diaphragm after the heart, is the most important muscle of the body." The diaphragm action also has an effect on blood pressure. During respiration the raised intra-abdominal pressure forces viscera against the inferior vena cava, blood is forced into the thorax, and blood pressure is raised, but falls again as inspiration ceases. Abdominal respiration causes an initial rise, followed by a fall in blood pressure during inspiration—and an initial fall followed by a rise in expiration acting against the changes induced by thoracic breathing. Hence the importance of this muscle with its rhythmical contraction, secondary to the heart alone.

In experimental work on dogs, Lemon studied the effect of abdominal incisions and of major surgical operations on the upper part of the abdomen to determine whether these really caused fixation of the diaphragm and contributed to the prevalence of postoperative pulmonary complications. His observations covered:

1. Simple incision in the abdominal skin.
2. Abdominal wall cut through and cavity opened.
3. The performance of an Eck fistula operation.
4. Eck fistula operation, plus removal of fractions of the liver.

The animals in his experiments were examined clinically and fluoroscopically immediately after operation, and each succeed-

ing day until the wound healed. In none of these animals was any fixation of the diaphragm discovered. He was unable to say whether this statement would hold true in regard to the diaphragmatic movement in man under simliar circumstances as no such experimental observation has been made in man. It is likewise admitted that no one can reproduce in the dog the conditions found in man which have been factors in the operation—the debility, anxiety, and apprehension, acuteness of sensibility to pain that lower vital capacity, and slow the venous stream in the abdominal veins. Lemon's impression is that the abolition of the resistance of the abdominal musculature against the downward thrust of the diaphragm has more to do with post-operative conditions than has been supposed. Likewise he felt that the dog did not lie on his back, that he was not trussed up with dressing, bandages, and encircling binders that would compress the margin of the ribs and prevent movement at the bases of the lungs when movement is required to prevent congestion. A low vital capacity in man is more an expression of abdominal pain preventing resistance to diaphragmatic contraction than to any loss of power in the efficiency of the diaphragm.

After unilateral phrenic neurectomy half of the diaphragm is paralyzed. It rises and is relatively immobile. The chest on the side of the neurectomy does not have a greater excursion in man and yet vital capacity returns to normal within a short time—nor does dyspnea follow exertion later.

This patient lost part of her diaphragm! The remainder was sutured down to a weak anchorage under increased tension and probably some temporary immobility. She lost a large proportion of the abdominal muscles and some of the ribs on the right side, which would act as resistance to the down thrust of the diaphragm, which would lower her vital capacity according to most observers. She had also all the shock of a major operation, involving removal of part of the liver—yet no postoperative pulmonary complication arose, even though the right lung was partly collapsed and a right pneumothorax existed at first after operation.

Following operation clinical examination revealed ordinary

breath sounds the second day on the right side. No early fluoroscopic examination of the diaphragm was possible. Later it was seen that the diaphragm made about its normal excursion and was somewhat below its normal level, where films made after two years showed it remained possibly permanently shortened a little.

The patient went on to active life in spite of a large abdominal hernia; did hard work, and bore three children since this operation.

This patient may furnish clinical confirmation in man: First, that the diaphragm has not a double enervation. Certainly a solitary enervation coming from the thoracic wall would have been largely destroyed in this patient. Second, that the lack of the resistance of the abdominal wall is not a great factor in postoperative pulmonary complication. Third, that the diaphragm is amenable to resection, to reinsertion, and seems to regather function as any other muscle.

TWO INSTANCES OF SMALL JOINT INFECTION IN ADULTS

THE next two patients illustrate acute destructive and probably metastatic lesions of the small joints in full-grown adults. These lesions are reasonably easy to overlook or to belittle and an intensive study of the two histories is worth while.

The first patient is W. P., male thirty-six years old, single, shipping clerk; was admitted to the Presbyterian Hospital 9/26/28 and discharged 9/29/28.

Diagnosis.—Destructive osteo-arthritis (acute) of metacarpophalangeal joint of right index finger.

The complaint was of pain and swelling in metacarpophalangeal joint of the right index finger of eight weeks' duration. He had a blister on the dorsum of this joint at the onset of the trouble. Then the hand, as a whole, began to swell and become painful, especially in this joint where the trouble had begun (Figs. 101–105). At the time of admission this joint was swollen, restricted in motion, painful to pressure, and all efforts to flex the right index finger. He could not make a fist with his finger. The skin over the joint was smooth and tense, but fluctuation was not definitely demonstrated. The previous history was negative in regard to infections. There had never been inflamed tonsils; he presented no glandular involvement. His weight is 152 pounds at present; ten years ago it was 174. He has been working indoors.

The family history was negative; two brothers died of typhoid.

A general physical examination exposed nothing abnormal. His blood pressure is 120/70 lying. Urine normal; Wassermann negative; blood, hemoglobin 80 per cent; white blood corpuscles 8700. Temperature on admission 98 F. An *x*-ray of the teeth was negative for root end infections. An *x*-ray examination showed partial destruction of first metacarpal head and the joint structures; also part of the proximal end of the proximal phalanx of this finger.

An operation was performed 9/26/28. Under ethylene anesthesia a Martin bandage was applied on the arm. A 2-inch incision was made on the medial aspect of the metacarpophalangeal joint of the right index finger. All tendons were avoided

Fig. 101.—Photograph showing the amount of swelling around the metacarpophalangeal joint of the first finger. This was acute, but the skin was not reddened. The function of the finger was seriously interfered with. There was no evidence of tendon sheath involvement.

by retraction, and the joint covering was exposed. These tissues were thickened and edematous; the joint seemed abnormally loose. When the joint was opened there was found a small amount of serosanguineous pus and liquid débris within it. The cartilage of the head of the metacarpal bone was nearly com-

pletely freed in one piece to lie loosely in joint. It was undermined and there was bony absorption of the head of the bone back for 1 cm. deforming the joint. The proximal surface of the proximal phalanx of the finger had undergone similar changes eroding the cartilage and joint surface. These joint surfaces were excised by a chisel and the thickened synovia lining the joint was dissected out as completely as possible. A culture was taken and the tissue saved for section and guinea-pig inoculation. The fibrous capsule of the joint was not disturbed except

Fig. 102.—Lateral view of the septic arthritis of the metacarpophalangeal joint of the index finger. Swelling and a slight glossiness of the skin is evident.

where it was opened on the lateral aspect. After the articular surface of the phalanx was excised a pocket extending $\frac{1}{2}$ cm. into the medullary portion of the bone filled with yellow soft débris was exposed and curetted out. The excised joint was swabbed with tincture of iodine; the capsule was closed; skin closed. A plaster-of-paris cuff was applied on the forearm and a banjo splint of heavy wire was fitted on so that traction in the long axis could be made on the index finger and maintained by rubber bands and tape. A slight postoperative fever as high as 99.8 F.

followed. A smear from the joint at operation showed polymorphonuclear leukocytes, no organisms; no acid fast bacilli were found. Cultures on blood agar, aerobic and anaerobic ascitic fluid, and broth cultures showed no growth. A guinea-pig inoculation was made 9/26/28. The pig was killed 11/3/28;

Fig. 103.—*x*-Ray film of the infection in the metacarpophalangeal joint of the index finger following resection of the joint. The two sesamoids and the flexor tendon of this finger are apparently seen through the aperture of the joint. The resected joint surfaces are not apposed and have been kept apart by traction. Lateral view shows the amount of separation and the relative cleanness of the bone ends.

the autopsy was negative for tuberculosis. The wound healed with some drainage and local reaction. Extension was kept on the finger eight days.

The patient has now 70 per cent normal range motion in the joint. *x*-Ray shows that the process in the bones is cured and a new joint has formed between the resected surface.

The second patient here is M. L., male, thirty-four years old, a cattle dealer, who was admitted to the Presbyterian Hospital 2/20/28. One week after the extraction of an abscessed tooth the present complaint began, consisting of pain and swelling in the left wrist which had lasted seven weeks at the time of admission. In the development of the complaint pain and

Fig. 104.—*x*-Ray of the distal radio-ulnar joint immediately after operation. Bone has been curetted away from the joint and granulations removed. There is a small amount of new bone formation seen around the outer side of the joint. Every effort was made at operation to avoid bursting through into the true radiocarpal joint. The carpal bones are here represented as of normal density.

swelling came on quite suddenly in the first interphalangeal joint of the middle right finger. The next day similar pain and swelling developed in the right wrist; two days later pain and swelling appeared in left wrist, which was severe, sharp, steady, aggravated by motion. He had no chills, fever, or sweats. There had been no recent loss of weight.

The patient's previous history was normal. He had had no

sicknesses. He was hard working, unmarried; had a good family history, and weighed 175 pounds. He possessed large tonsils with some white material in the crypts, and two remaining carious teeth which were yet to be extracted.

A general physical examination showed no abnormalities. His left wrist was swollen; looked at on both the dorsal and volar aspect. Great pain was caused on either active or passive motion

Fig. 105.—This *x*-ray film shows the condition of the distal radio-ulnar joint four months after it was thoroughly cleansed and drained; new bone is filling in the space and the joint surface seems to be partially obliterated. The changes in the carpal and metacarpal bones are those of atrophy of disuse, as there is no evidence seen that these joints were infected or that the bones were involved in the extension of the process.

and pressure over the distal radio-ulnar joint was very painful. The whole hand was now swollen and tense. No redness was seen but the swelling extended about 2 inches up the forearm, especially on the dorsum; no crepitus was noticed in the wrist movements and all hand and wrist motions were restricted by pain. His temperature was 99 F. on admission; after operation it rose to 100.2 F.; after one day it fell to normal and remained

there. His blood findings on admission were white blood cells 11,000; hemoglobin 85 per cent; urine normal. The blood pressure readings were 132/80.

An *x*-ray examination of the left wrist showed an area of destruction at the distal end of the radius and ulna starting in the distal radio-ulnar joint extending into the diaphysis of each bone at the point of mutual contact. There is a little subperiosteal thickening above this on both the radius and ulna. A moderate amount of bone atrophy was present in the shafts of the two bones.

Operation was performed 2/21/28 under ethylene anesthesia after the application of a Martin bandage. An incision 7 inches long was made on the outer border of the left ulna; the only tendon identified was the flexor carpi ulnaris. All soft parts were retracted away from the ulna and the distal radio-ulnar articulation was exposed. This was filled with granulations and remnants of ligamentous structures. The ulna was quite free from its attachment to the radius but was not definitely dislocated. The necrotic bone surfaces and the area surrounding this joint was curetted and rendered mechanically clean. A capillary drain was inserted extending out laterally; the soft parts partly closed. The patient's hand was put in a cock-up position and a molded plaster-of-paris splint was applied.

Postoperative Course.—Considerable bloody discharge followed the operation; after three days the capillary drain was removed. One week after operation all swelling in the fingers was gone; the immobilized wrist was not painful; the wound no longer discharged. Ten days after operation the patient was discharged from the hospital still wearing the splint.

x-Ray examination on 2/28/29 showed in the film of the left wrist a separation between lower end of radius and ulna and some evidence of operative procedure as the bone margins are now sharply defined. Some new bone formation in this area and a little periosteal thickening along the radius and ulna were present and the *x*-ray evidence pointed to a filling in rather than an extension of the process.

The pathologic diagnosis was chronic inflammatory tissue and edema; no evidence of tuberculosis; cultures all negative.

Follow-up History.—4/12/28: Much soreness in left wrist with some swelling. *x*-Ray shows possible involvement of radio-carpal joint and beginning disintegration of navicular. Left forearm, wrist, and hand put in circular plaster dressing for complete immobilization.

6/26/28: *x*-Ray shows anteroposterior and lateral views of left wrist with increase in bone on mesial side of distal end of radius. Carpal bones show a good deal of atrophy as compared to films of 2/28/28. The splint was removed at this time and use of the hand was started. His wrist motions today are 80 per cent normal, his finger motions are complete and the hand grasp and power are approximately what they have been for years.

Several factors may have had to do with the satisfactory results obtained with these two patients. From an etiologic standpoint the infections were not very mild, they produced fever, much pain, leukocytosis along with bone and joint destruction. They were possibly a little more severe than many of the joint infections which develop into what is called chronic osteo-arthritis with permanent bone and joint changes; they certainly were more extensive than the so-called "rheumatoid infections of joints." However, wanting any proof of etiologic factor in many instances of arthritis and osteo-arthritis it appears that these two clear-cut small joint infections may throw some light on those of lesser degree which afflict so many individuals. There is a great leaning toward the bacterial causes of "rheumatic" joints, the biochemical causes holding fewer supporters year by year.

From a surgical standpoint we may argue that these two patients illustrate the necessity for a careful search by *x*-ray of all small joint disturbances. Either case might have been considered tuberculosis clinically, but the boldness of the surgical attack not only aims to settle the diagnosis but to limit the destructive process, to stop the pain and eventually to restore all function it is possible to save. A thorough exposure of the joint involved avoiding any further damage to adjacent structures, complete eradication of the pathologic cartilage and bone, preservation of the capsule of the joint and adequate splinting, traction, and follow-up care promise the greatest functional return in similar joint infections.

CLINIC OF DRS. PERCIVAL BAILEY AND
PAUL C. BUCY[1]

FROM THE DEPARTMENT OF SURGERY, UNIVERSITY OF CHICAGO

TUMORS OF THE SPINAL CANAL

WE are presenting a group of 7 cases, each with a different type of involvement of the spinal cord, for the purpose of considering the diagnostic problems which they present and the results of surgical interference.

NEUROFIBROMA OF THE CERVICAL CORD

This young patient has an unusually interesting condition in that we find evidence of several forms of involvement of the central nervous system which occur in von Recklinghausen's disease, or generalized neurofibromatosis.

Case I. Pain in Back of Head and Neck. Marked Weakness and Atrophy of Neck Muscles. Weakness in Upper Extremities. Spastic Paraplegia. Cutaneous Neurofibroma. Intracranial Calcification. Choked Disks. Laminectomy with Removal of Neurofibroma Anterior to Medulla and Cervical Cord. Marked Improvement.—J. L. of Chicago, male, twelve years of age, was referred to this clinic by Dr. Clark O. Melick of Chicago.

He was admitted on April 9, 1929 complaining of inability to walk, weakness in his arms, and pain in the back of his head and neck. Two years prior to admission he noted weakness in his right arm and was unable to raise his hand to his mouth. About the same time he began having severe pains in the occipital region. His neck became very stiff and movement of it caused pain. The pain in the occipital region occurred chiefly

[1] Clinic given before the American College of Surgeons, October, 15, 1929.

in the morning and could be relieved by standing upright. However, the pains would also occur at other times if he lay too long in one position. About a month later he noted that his right leg began to drag, and his playmates observed that he was easily upset and frequently devised means of tripping him. In September, 1927, he was admitted to another hospital. Here the weakness in the right arm was noted, the increased knee jerks and bilateral positive Babinski. *x*-Ray examination revealed what was thought to be a congenital abnormality of the third cervical vertebra. There was a slight choking of the optic disks and the patient had been observed to have momentary attacks, while walking, of tonic spasms in which his arms would be extended and he would rise on his toes. He gradually passed into a semiconscious state with arms and legs rigid and extended. As an intracranial lesion was suspected a left subtemporal decompression was performed but no lesion and no increased intracranial tension was found. *x*-Rays of the skull taken after the operation were considered normal. Subsequently the patient improved considerably; he again was able to be up and about. However, before very long his legs began to become stiff and the tonic spasms returned. His condition varied greatly from time to time, but for a month prior to admission he had been unable to walk.

Examination.—His general physical condition was good. A pulsating, but not bulging, left subtemporal decompression was present. A small, hard nodule in the skin about 1.2 cm. in diameter directly over the spine of the fifth dorsal vertebra was the only peripheral manifestation of von Recklinghausen's disease. Ophthalmoscopic examination revealed a choked disk of 3 diopters in the right eye and 2 in the left with atrophy. The visual fields were generally constricted. The jaw deviated markedly to the left and the left sternocleidomastoid muscle was stronger than the right; otherwise the cranial nerves were normal. Pressure over the cervical vertebrae elicited pain as did flexion of the head on the chest. There was marked weakness in all muscles of the neck and the patient was unable to lift his head from the bed. There was considerable weakness in

both upper extremities, more marked on the left, and particularly noticeable in the movements of the fingers and wrists. There was also very marked weakness in all muscles of the lower extremities, somewhat more on the left. There was some increase in tone in the arms and the legs were very spastic. Defense reflexes were present though no definite upper level of elicitation was determined. Sensory examination revealed a hypesthesia to pain, touch, and temperature in the third and fourth cervical dermatomes only, especially on the left side.

The deep reflexes were active in the upper extremities, greater on the left than on the right. The abdominal and cremasteric reflexes were practically absent. Patellar clonus and ankle clonus were present on both sides. Babinski's sign was positive bilaterally.

A lumbar puncture was done. The initial pressure was 110 mm. of fluid. It rose slowly to 180 on jugular compression. The spinal fluid was yellow in color. The total protein was 204 mg. per 100 c.c. of fluid.

Roentgenograms.—The *x*-rays of the skull revealed a small calcified mass in the right parietal region.

Lipiodol injected by lumbar puncture was observed under the fluoroscope. With the patient tilted, head down, the lipiodol was observed to flow freely through the lumbar and lower dorsal regions until it reached the level of the sixth dorsal vertebra where it hesitated and then divided into two thin streams, one on either side of the spinal canal. It then slowly passed this point, passing into the cervical region to the lower level of the third cervical vertebra, where it was completely arrested. *x*-Rays of the cervical spine seemed to indicate a slight widening of the intervertebral space on the right side between the second and third cervical vertebrae.

Diagnosis.—A diagnosis was made of multiple neurofibromatosis, with glioma of the optic chiasm, calcified tumor in the right parietal region, possibly in the choroid plexus, neurofibromata in the spinal canal at the third cervical and sixth dorsal vertebrae and a neurofibroma of the skin at the level of the fifth dorsal spine.

Operation.—On April 15, 1929 a laminectomy was done removing the laminae of the first, second, third, and fourth cervical vertebrae and a considerable portion of the occipital bone about the foramen magnum. When the dura mater was incised the cervical cord above the fourth vertebra was seen to be very wide, filling the entire canal, grayish in color and quite avascular. At first it was thought that we were dealing with an intramedullary tumor. However, after considerable difficulty the cord was rotated slightly exposing an extramedullary tumor anterior to it. In order more completely to expose the tumor it was necessary to cut the first, second, and third cervical roots on the left side. A large tumor extending from the fourth cervical vertebra into the cranial cavity beneath the medulla was removed in fragments. It was then evident that the tumor penetrated the dura mater and extended outward through the second intervertebral foramen to form another tumor 3 x 2 cm. in diameter, a typical "hour-glass" tumor. The extradural portion was also removed.

Postoperative Course.—The patient improved rapidly. For the first four days after operation the subtemporal decompression bulged markedly. Within a week the spasticity was definitely less and motion in the lower extremities was greatly improved. Within twenty-six days after the operation he was able to walk without support. The spasticity had practically disappeared. Reflexes were almost normal and the positive Babinski was no longer present. However, there was an area of total anesthesia in the first, second, and third dermatomes on the left and the patient held his head to the right, both due to the root sections done during the operation.

Since his discharge from the hospital he has been seen at frequent intervals. His improvement has been very rapid and at present except for the tilting of the head to the right, he has practically completely recovered.

Pathology.—Sections of the tumor removed at operation reveal it to be a typical neurofibroma. The tissue is composed of elongated spindle-shaped cells arranged in bundles. The nuclei assume the typical palisade formations (Fig. 106). The

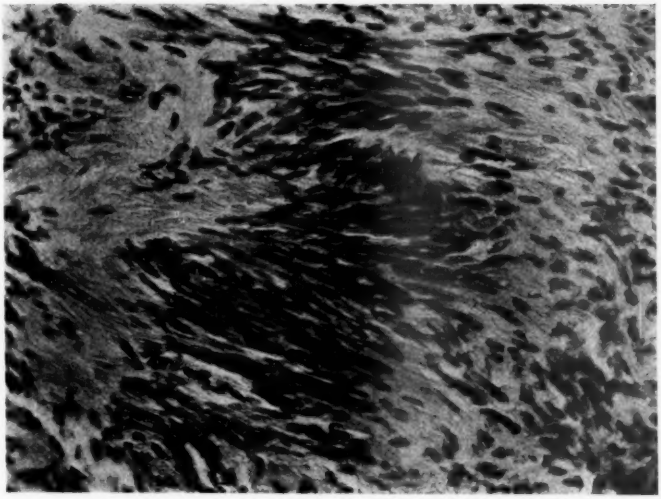

Fig. 106.—Case I. Neurofibroma in the spinal canal. The elongated cells with their oval nuclei arranged in palisade formation. Hematoxylin and eosin (× 100).

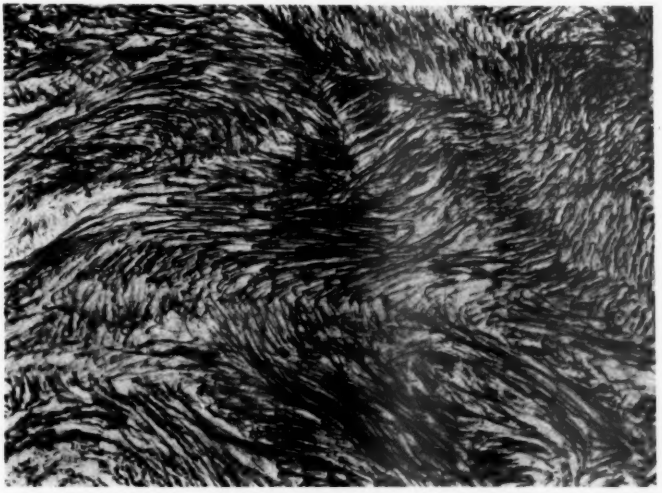

Fig. 107.—Case I. Neurofibroma. The great mass of reticulin fibers which lie between the individual cells of the tumor. Perdrau's silver impregnation (× 100).

silver impregnations by Perdrau's method reveal enormous amounts of reticulin between the cells (Fig. 107).

Discussion.—The diagnosis of the lesion which produced the major portion of this patient's symptoms was complicated by the multiplicity of the foci. The presence of the choked disks, a calcification possibly in the choroid plexus, and attacks of what appear to be decerebrate rigidity undoubtedly led to the first error and the performing of a subtemporal decompression. However, in view of that error and with the positive cerebrospinal fluid findings of a partial block, xanthochromia and an increased protein content, we were able to make a diagnosis of a tumor of the spinal canal. The localization of this lesion in turn was complicated by the evidence, from lipiodol injection, of two tumors within the spinal canal. However, we chose to operate on the one in the cervical region because of the involvement of the upper extremities, as well as the lower. The presence of the cutaneous nodule led us to the correct interpretation of the lesion as a neurofibroma.

It is of interest to consider here the presence of the choked disks in this case. In the absence of any intracranial tension as demonstrated by the flat, pulsating decompression, one must postulate a lesion in the neighborhood of the optic tracts themselves to account for this phenomenon. The most likely lesion in this region in a case of neurofibromatosis is a glioma of the optic chiasm of the type known as spongioblastoma (unipolare et bipolare). During the past year we have observed several such cases in this clinic. Though it is uncommon for them to have other lesions in the central nervous system, it has been recognized for many years that cases of von Recklinghausen's disease sometimes become blind, but it has not been so commonly realized that the loss of vision is due to a glioma of the chiasm.

It is difficult to say what the future of this boy will be. Certainly there are several possibilities. The lesion of the optic chiasm may progress to the point where vision is completely lost. Already a generalized constriction of the visual fields has occurred. Fortunately these tumors grow very slowly, as a rule, and seem to respond well to *x*-ray therapy.

The remaining tumor in the spinal canal at the sixth dorsal vertebra may, at some later date, become of sufficient size to compress the cord and produce symptoms below that level. It is unlikely that the other intracranial lesion, providing it is only a calcification of the choroid plexus, will cause any trouble. Such lesions are very frequent, but rarely the source of any symptoms.

Neurofibroma of the spinal canal of "hour-glass" shape is quite common and must always be borne in mind in dealing with this type of lesion. It is very easy to remove either the intradural or extradural portion and overlook the remaining tumor, thus leaving an excellent source for a recurrence which will necessitate a second operation. Many such instances have recently been recorded by Heuer.

MENINGIOMA OF THE CERVICAL CORD

This case is very similar in its location and symptomatology to the preceding one.

Case II. Pregnancy. Gradually Progressive Quadriplegia. Laminectomy. Meningioma. Complete Recovery.—A. C., a white female, twenty-six years of age, was referred to this clinic by Dr. G. W. Hall of Chicago.

The patient entered the hospital on January 19, 1929, complaining of paralysis of all four extremities and numbness of her entire body. She stated that she menstruated last in March, 1928. Her pregnancy was uneventful until July, when she began to suffer from numbness and tingling in the ulnar aspect of both forearms and hands. About the same time she noted beginning weakness in the upper extremities and shortly afterward the legs became involved. Rather marked constipation had been present since the spring of 1928, but had been attributed to the pregnancy. The weakness in both her arms and legs gradually became worse until in December she was unable to walk. About the same time she began to require enemas and had to be catheterized. Shortly afterward she noted a sensation about her chest as though a tight band were constricting it.

She was seen by Dr. Hall, who diagnosed a spinal-cord tumor and on December 31, 1928 a cesarean section was done by Dr. Gough at St. Luke's Hospital, without anesthesia of any kind.

Examination.—When examined here on January 19, 1929, there was marked weakness in the upper extremities and definite atrophy of the small muscles of the hands. There was also marked weakness of the legs with considerable spasticity. Defense reflexes could be elicited up to the level of the second rib.

Sensory examination revealed decreased sensation to painful, thermal, and tactile stimuli up to the level of the second rib in front, the second thoracic vertebra posteriorly, and over the arms up to the shoulders.

The third and fourth cervical spines were tender to pressure. All deep tendon reflexes were exaggerated. The abdominal reflexes were absent. There was bilateral ankle clonus and positive Babinski's signs.

Fluoroscopic examination revealed marked limitation of motion of the right side of the diaphragm. Lumbar puncture at St. Luke's Hospital had revealed a typical Froin syndrome (yellow fluid which coagulated, and high protein content) and a complete block of the cerebrospinal fluid.

Diagnosis.—A diagnosis of tumor in the upper cervical region was made.

Operation.—On January 23, 1929 a laminectomy was performed. On incising the dura mater a large grayish-brown tumor was seen lying posterior and to the right of the cord at the level of the second, third, and fourth cervical vertebrae. The tumor and the meninges at its point of attachment were readily removed.

Postoperative Course.—The patient had to be catheterized for a few days, but within a week's time she was able to move her arms much better, and eighteen days after operation she was able to walk with support. Although spasticity was still present, it had definitely diminished. During the next month her progress was quite gradual, but at the end of that time she was able to walk well without support.

When last seen on October 15, 1929 she was in perfect health, completely recovered from all her symptoms and findings.

Microscopic Description.—Sections of the tumor removed at operation showed it to be a typical meningioma of the psammoma type (Fig. 108). The cells were arranged in bands and whorls and at the center of the latter appeared many strands of hyaline-like material which, in many instances, was calcified; typical psammoma bodies.

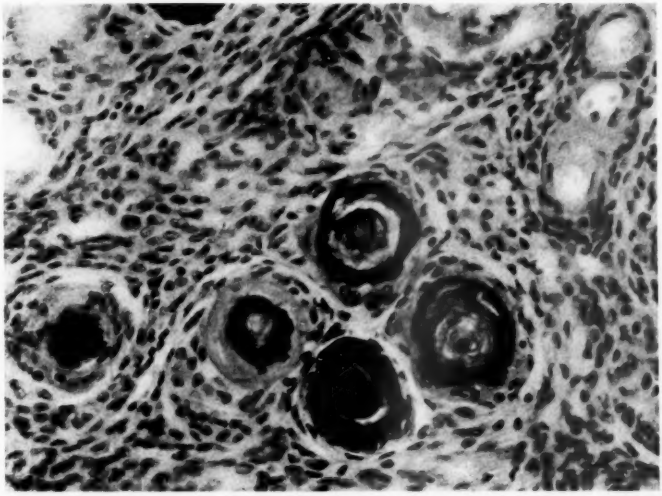

Fig. 108.—Case II. Meningioma of the spinal canal. The whorllike arrangement of the cells with collagen formation in them and the deposition of calcium salts (psammoma bodies) is typical of this type of tumor. Hematoxylin and eosin (× 100).

Discussion.—This case is interesting for many reasons. First, it emphasizes the factor of pregnancy as a precipitating cause, both in tumors of the spinal cord and of the brain.

The presence of the atrophy of the small muscles of the hands is also of considerable interest. It is definite evidence of involvement of the anterior horn cells of the lower cervical cord, but does not necessarily mean direct involvement of this area by the tumor. In such cases as this, it must be due to involvement of the blood supply of the cord by a tumor which lies at a higher level.

The sensory involvement of the arms, the paresis of the diaphragm, and the tenderness of the third and fourth cervical spines established the localization of the lesion.

CIRCUMSCRIBED ARACHNOIDITIS

We are dealing here with a condition which is often difficult to distinguish clinically from tumor.

Case III. Spastic Paraplègia of Two Years' Duration. Laminectomy. Circumscribed Arachnoiditis. Improved.—D. B., of Chicago, Illinois, female, eleven years of age, was referred to this clinic by Dr. G. B. Hassin of Chicago.

She was admitted on October 1, 1929 complaining of inability to walk. About two years prior to admission it was noted that her left foot became markedly inverted and that she walked on the side of the foot. This increased in severity and a short time later a cast was applied. When this was removed six months later she was unable to walk without assistance. This condition continued unchanged for about a year. There have been no sensory manifestations nor any difficulty with either bowel or bladder. A year prior to admission a lumbar puncture had been done at another hospital following which the paralysis became much more marked.

Past History.—Of interest is the fact that at the age of four, five years prior to the onset of symptoms, she fell from a height of 20 feet, alighting on her buttocks. There was no evidence of serious injury at the time.

Examination.—When examined on admission the general physical condition was found to be good. Neurological examination revealed sensation to be normal in all phases. There was a positive Beevor sign. Both lower extremities were exceedingly spastic in extension. She was able to walk very poorly with support. The strength in the extensors of the legs was quite good, while the flexors were very weak. The same was true of the musculature of the feet. The upper abdominal reflexes were active, the lower were very weak. The patellar

and Achilles' tendon reflexes were very markedly exaggerated. There was a positive Babinski sign on either side. Defense reflexes were obtained up to the level of the iliac crests on either side. There was no sensory loss.

A lumbar puncture was performed and revealed a partial block of the cerebrospinal fluid pathway. The fluid was clear and colorless and the total protein was only 81.6 mg. per 100 cc. of fluid. The test for globulin gave negative results. *x*-Ray examination of the spine revealed no lesion.

Diagnosis.—A diagnosis of compression of the spinal cord at the level of the seventh and eighth thoracic vertebrae was made.

Operation.—On October 8th, a laminectomy was performed, the laminae of the seventh, eighth, and ninth thoracic vertebrae being removed. The dura mater was found to be markedly adherent to the arachnoid and the arachnoid itself greatly thickened and grayish in color. There was a dense band of tissue, about 4 mm. thick, stretching from the dorsal surface of the cord to the dura mater at the lower level of the seventh vertebra. These adhesions were filled with fatty tissue. The pia mater was also very much thickened and this was split over the dorsal surface of the cord, revealing the latter to be flattened and yellow in appearance.

Postoperative Progress.—Within a week after operation she was able to move her legs much more freely; they were less spastic, although the reflexes were unchanged. She was discharged twenty-five days after operation very much improved. She was able to walk much better, although still requiring support. The spasticity of her lower extremities was much decreased. The reflexes were still hyperactive but not nearly to such a great degree as before operation. Bowel and bladder functions were still normal and there were no sensory changes. When seen a month later she was able to walk for short distances and the spasticity had practically disappeared.

Microscopic Description.—Examination of the small band of tissue removed at operation revealed it to be composed largely of large fat cells lying in a mesh of loose connective tissue.

Discussion.—Circumscribed arachnoiditis of the spinal cord has been ascribed to three main causes: trauma, syphilis, and severe infections of the meninges. Recently 4 cases have come under our observation. The first was very extensive and associated with a hypertrophic spinal arthritis. The second occurred in a patient who died of carcinoma of the pancreas and in which there was no other obvious etiology. The third is the patient under discussion and the fourth occurred in a woman who had several years before suffered from a severe typhoid-meningitis. In the present patient the only factor to which we could attribute this condition was the fall which she had five years prior to the onset of this illness. In this connection it is of interest to note that the two patients with the more marked lesions had both sensory and motor changes while the other two had only motor involvement. Because it is possible in this condition to have only the signs of a spastic paraplegia, and as it may be on a luetic basis, every case of so-called Erb's spastic paraplegia should be carefully investigated as the symptoms may be due to arachnoidal involvement and therefore amenable to surgical treatment.

This patient is of particular interest because of the ease with which the lesion was localized in the presence of only motor manifestations. Beevor's sign (the drawing upward of the umbilicus when the head is elevated) indicates that the abdominal muscles below the umbilicus are paralyzed while those above are intact, and therefore when they contract draw the umbilicus upward. This finding was further corroborated by two others. The upper abdominal reflexes were active, the lower were absent; and the defense reflexes were present almost up to the umbilicus. Many years ago Babinski pointed out that in a complete spastic paraplegia the defense reflexes were present up to the lower level of the lesion. Thus as the umbilicus is the junction of the ninth and tenth thoracic dermatomes we knew that the lesion was at the ninth and tenth dorsal segments of the spinal cord and lay at the level of the seventh and eighth dorsal vertebrae.

This case is of interest because of the sensory findings.

Case IV. Urinary Retention. Rapidly Developing Spastic Paraplegia with Dissociation of Sensation. Laminectomy. Extradural Sarcoma. *x*-Ray Therapy. Improvement.—S. B. of Canton, Ohio, a white male sixty-three years of age was referred to the Albert Merritt Billings Hospital by Drs. G. B. Hassin and L. L. Charpier of Chicago.

He was admitted to the hospital on October 8, 1929 complaining of paralysis of his lower extremities, numbness in his legs and pain in the lumbosacral region. He stated that about April 1, 1929 he began having pain in the lower part of his back which radiated around into his lower abdomen. The pain varied in intensity and was aggravated by lying down. For several months previous he had noted slight difficulty in urinating but it had not particularly attracted his attention until on July 4, 1929 he was unable to urinate at all. At that time he was catheterized and a large quantity of bloody urine obtained. Subsequently it was always necessary to catheterize him until on July 16th a perineal prostatectomy was performed. The pathologic diagnosis was benign adenoma of the prostate. He made an excellent postoperative recovery and was free from symptoms until the latter part of August when the pain in the back again returned. At this time it was situated just beneath the costal margin. However, he continued to gain in weight and strength and on September 16th returned to his work in a steel mill. A few days prior to returning to work he had noted a slight stiffness in his knees and on the 16th numbness was present in his legs below the knees. The numbness became more severe but did not extend, and he began to have difficulty in walking. He stopped work on the 20th. On September 22d his legs had become still weaker and he required a cane for support in walking. The following day they were still weaker and on the 24th he was unable to walk at all. About this time he began to have a sense of constriction about the lower abdomen and during the first week of October the numbness extended up his thighs to the groin.

Examination.—When examined on October 9, 1929 the patient was completely paralyzed in his lower extremities except for very slight movements in his toes. Defense reflexes were present but no definite level could be determined in this manner. All abdominal reflexes were absent except the left epigastric which was very weak. The cremasteric reflexes were also absent. The deep tendon reflexes were markedly hyperactive in both lower extremities. A persistent ankle clonus was present bilaterally. Both Babinski's and Oppenheim's tests elicited dorsoflexion of the great toes.

Sensory examination revealed tactile sensation to be intact everywhere. Pain and thermal sensations were diminished from the ninth dorsal to the first lumbar dermatomes and completely absent below the first lumbar level; the upper level was not sharp.

A lumbar puncture was done. The initial pressure was 110 mm. of fluid and it failed to rise on compression of the jugular veins indicating a complete block of the spinal subarachnoid space. The spinal fluid was slightly yellow and only 4 cc. could be obtained. The total protein content of the spinal fluid was 201 mg. per 100 cc. of fluid. The examination of the fluid was otherwise negative.

Roentgenograms.—Roentgenological examination of the vertebral column was entirely negative. However, at the time of lumbar puncture $1\frac{1}{2}$ cc. of lipiodol was injected into the subarachnoid space and with the patient on the tilting fluoroscope, with his head down, the lipiodol was seen to stop abruptly at the level of the inferior surface of the eleventh dorsal vertebra.

Diagnosis.—A diagnosis of spinal cord tumor extending from the seventh to the eleventh dorsal vertebrae was made.

Operation.—A laminectomy was performed on October 12th, the laminae of the seventh to the eleventh thoracic vertebrae being removed. It was immediately evident that the usual peridural tissues had been replaced by a tough reddish-gray mass which completely surrounded the spinal cord outside of the dura and extended practically the whole length of the incision. It was impossible entirely to remove this tumor, which was quite

vascular, but the dorsal portion was split and as much as possible removed.

Postoperative Progress.—It was necessary to catheterize the patient for about two weeks after the operation. He soon began having pains in his feet and ankles, but showed no improvement in the paralysis. On October 26th, fourteen days postoperative, deep *x*-ray therapy to the involved area was started. In the course of the next six days he was given an erythema dose

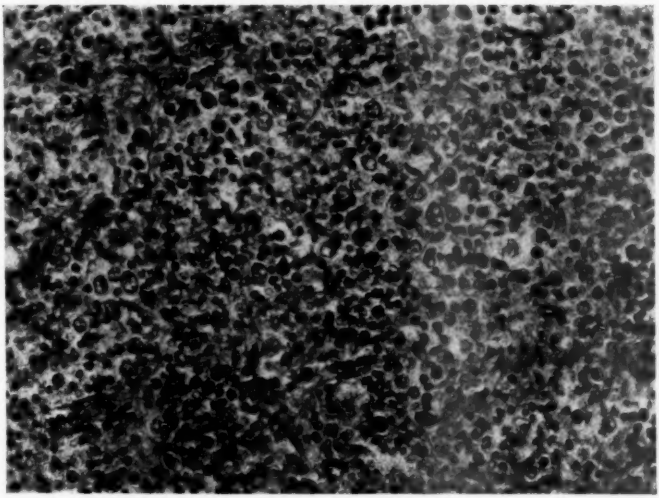

Fig. 109.—Case IV. Extradural sarcoma. The tumor is extremely cellular and composed of cells with round nuclei varying somewhat in size and having an indefinite cytoplasm. Hematoxylin and eosin (× 100).

(550 "R" units). On October 28th he began complaining of involuntary jerking of his legs and two days later was able to move his right leg. On November 3d, he was able to move both legs and when discharged on November 5, 1929 he was able to sit up in a chair, the strength in his legs was rapidly increasing, sensation had returned to some degree over his lower extremities and he was having no trouble with his bladder. By November 21st he was able to walk without support and the spasticity had almost disappeared.

Microscopical Description.—The small piece of tissue removed at operation revealed a typical undifferentiated small round-cell sarcoma (Fig. 109).

Discussion.—Because of the rapid onset of the paralysis and sensory loss in this case we at once thought of the possibility of a metastasis from a malignancy of the prostate. However, on inquiring of his former physicians we were told that the part removed showed no evidence of malignancy. Moreover, because of the dissociation of sensation (the absence of pain and thermal sense in the presence of an intact tactile sensation) a tumor within the cord itself seemed most probable. Such a dissociation of sensation has long been considered evidence of an intramedullary lesion, particularly syringomyelia, and it is of interest to know that it can also be produced by compression of the cord from a lesion outside of it.

It is important to observe in this case that improvement occurred only after the administration of *x*-ray therapy as it indicates the value of such treatment in this type of case. Of course this treatment will be continued.

INTRAMEDULLARY TUMOR

The following case is typical of tumor in this location.

Case V. Gradually Progressive Quadriplegia. Lumbar Puncture with Xanthochromic Fluid and Complete Block. Laminectomy. Intramedullary Tumor. Slight Gradual Improvement.—E. K. of Atlantic, Iowa, a white boy fifteen years of age was referred to this clinic by Drs. W. P. Coons and G. W. Hall of Chicago.

He was admitted to the hospital on June 23, 1929 complaining of inability to use his legs and marked weakness in his hands and arms.

In November, 1928, he had noticed that his left leg "did not respond" as well as his right and that in walking through the corn stubble he would catch his left toe. In December his parents noted that he dragged his left foot. The paresis of the left lower extremity became progressively worse and in April,

1929 the little finger on the left hand became swollen and in a few days this extended to the entire hand, and he had difficulty in using it. However, this condition was not constant until a month later when the hand became swollen, cold and cyanotic, and remained so. About a week prior to his admission in June the right hand also became awkward but was never as bad as the left. On June 21, 1929, two days before admission here a lumbar puncture was done by Dr. G. W. Hall. This revealed a complete block and xanthochromic fluid. Subsequently the right leg became stiff and all of the other symptoms were exaggerated. Marked constipation and difficulty in urinating had been present since February, 1929. At no time had the patient suffered any pain.

Past History.—Of interest in his history is the fact that about five years prior to the onset of the difficulty in walking, the "cords of his neck on both sides became stiff and tender" and three years later while getting on a horse over its head the horse suddenly raised its head striking him under the chin and throwing him to the ground. He was not unconscious but was "dazed" for two days.

Examination.—June 23, 1929. The left hand and arm were red and cold and somewhat swollen. There was some atrophy of the muscles of the hands and forearms and marked flaccidity. The loss of strength was extremely great, being slightly more marked on the left. The deep tendon reflexes were greatly diminished in the arms. The flexors of the neck were also very weak while the extensors and rotators were much stronger. Fine fibrillary twitchings were present in the trapezius muscles. The abdominal and cremasteric reflexes were absent.

The lower extremities were very spastic, the left more than the right. There was also a marked loss of strength especially in the flexor groups. Defense reflexes were very active but no definite upper level was determined. The deep tendon reflexes were very hyperactive. A sustained ankle clonus was present on both sides and Babinski's sign was present bilaterally.

Sensory examination revealed a marked hypesthesia to touch up to the second rib anteriorly and the second thoracic vertebra

posteriorly, also over the arms. There was also a marked hypothermesthesia and hypalgesia over the same area, but more marked on the right side, the involvement of temperature sense being greater than that of either pain or tactile. Examination of the pupils revealed no sympathetic involvement. There were numerous widely scattered cutaneous pigmented moles.

Laboratory.—Examination of the spinal fluid removed by Dr. Hall revealed a total protein of 209 mg. per 100 cc. The fluid was yellow in color and coagulated on standing. Roentgenological examination revealed a normal cervical spine and under the fluoroscope the diaphragm was seen to move normally.

Diagnosis.—A diagnosis of spinal cord tumor in the lower cervical region was made.

Operation.—On June 25, 1929 a laminectomy was performed removing the laminae of all cervical vertebrae and a portion of the occipital bone about the foramen magnum. On incising the dura mater the cord bulged through the opening and when completely exposed the cord was seen to be very greatly enlarged, completely filling the spinal canal. It was soft and grayish in color. The lesion extended upward involving the lower part of the medulla oblongata. No effort was made to remove the tumor.

Postoperative Course.—Following the operation the patient's condition was much the same except that the red, cold, swollen condition of the left hand diminished. A week after the operation he was able to use his hands a little better than before. The hypothermesthesia, hypesthesia, and hypalgesia now involved all of the body except the face. He was discharged two weeks after the operation and returned to his home.

We were much surprised when on September 22d we received a letter from his mother stating that he had made progressive improvement since arriving home. At that time he was able to sit up and had been out riding in an automobile. He also was able to use a typewriter and could move his legs much better than before operation.

Discussion.—This boy presented signs of involvement of almost all of the structures of the cervical cord. The spastic

paraplegia indicated the involvement of the lateral columns; the hypesthesia to touch involvement of the dorsal columns; while the weakness, atrophy, fibrillary twitchings, and diminished reflexes of the upper extremity and shoulder girdle were definite evidence that the anterior horn cells were also encroached upon by the lesion.

The vasomotor change in the hands was typical of intramedullary autonomic involvement and is seen not only in intramedullary tumors but is even more frequent in syringomyelia, but the major involvement of the musculature on the left side and of pain and thermal sensation on the right side, known as the Brown-Séquard syndrome, indicated that the lesion might be extramedullary and pressing on the left side of the cord. We were the more inclined to the latter diagnosis because of the presence of numerous cutaneous pigmented moles.

The lumbar puncture with a typical Froin syndrome (yellow fluid, coagulation of the fluid, a high protein content) and a complete block of the cerebrospinal fluid pathway gave definite evidence that the lesion was a tumor.

The ultimate outcome of such a case is obvious. The neoplasm, extending as it did into the medulla oblongata, was far too extensive to permit of surgical removal.

TUMORS OF CONUS MEDULLARIS AND CAUDA EQUINA

The next 2 cases are typical examples of tumors in this location. The first one involved the conus medullaris and the upper part of the cauda equina; the second lay farther down the canal. Clinical methods for differentiating lesions of the conus from those of the cauda are frequently given but actual experience will soon demonstrate their uncertainty. The great similarity of the 2 cases presented here is obvious. It is in tumors in this location that the use of lipiodol is of the most value. We are aware that considerable criticism of the use of lipiodol intraspinally has recently appeared, but do not believe that it is compatible with clinical experience. We have used it in many cases in this clinic and have yet to see any untoward results even of a temporary nature. However, we do not advo-

cate its use in every or even a majority of the cases. Certainly all of the usual neurological tests should be utilized before resorting to this mechanical means.

Case VI. Pain in Lumbar Region Extending Down Right Leg. Partial Block of Cerebrospinal Fluid. Localization with Lipiodol. Laminectomy. Tumor of Conus Medullaris and Cauda Equina. Marked Improvement.—J. W., a white male, fifty-four years of age, was referred to this clinic by Dr. Peter Bassoe of Chicago.

He was admitted on April 30, 1929, complaining of pain in his back radiating down the right leg. He stated that two years prior to admission he began suffering with pain in the lower part of the back. There was no radiation of pain at this time. A diagnosis of sciatica was made and his teeth were removed. He recovered completely and remained free from pain until nine weeks before his entrance to this hospital, when the pain in the back recurred and was more severe on the right side. The pains occurred mostly at night. About three weeks later the pains began to radiate down the back of the left leg and down the right leg both anteriorly and posteriorly. He began to have some difficulty in urinating and slight incontinence. Coughing or sneezing greatly aggravated the pain. On April 25th a lumbar puncture was done elsewhere. Subsequently, the pain was much worse. At no time did he notice any weakness in his legs, nor any numbness.

Examination.—Station and gait were both normal. There was no demonstrable weakness. The abdominal reflexes were active and equal. The knee jerks were very weak. The Achilles' tendon reflexes were more nearly normal, but were less active on the right side. Babinski's sign was not positive.

Sensory examination revealed an area of hyperesthesia over the anterior surface of both thighs, especially the right and a diminution in tactile sensation in the perianal region, dermatomes of the fourth and fifth sacral segments.

A lumbar puncture was done revealing an almost complete block of the cerebrospinal fluid. The fluid was very yellow and coagulated on standing. The Ross-Jones test for globulin was

strongly positive. About forty-five minutes after the puncture he developed excruciating pain down the back of the right thigh.

Lipiodol was injected through the lumbar puncture needle and the patient examined under the fluoroscope. With the patient tilted, head downward, the lipiodol flowed to the level of the middle of the second lumbar vertebra where it was completely arrested.

Diagnosis.—A diagnosis of tumor of the conus medullaris and cauda equina was made.

Operation.—On May 4, 1929 a laminectomy was done, removing the laminae of the first, second, and third lumbar vertebrae. The dura bulged backward and was dark in color. On incising it a dark grayish-red tumor mass lying dorsal to the roots of the cauda could be seen. The tumor was firmly adherent to the nerve roots on the right side. The capsule was incised along its entire dorsal surface and the contents removed. Then as much of the capsule as possible was cut away.

Postoperative Course.—It was necessary to catheterize the patient for eleven days after the operation. Otherwise his convalescence was uneventful. He was up in a chair on May 15th. Deep *x*-ray therapy was started on May 17th. He was discharged on the 18th and returned on the 20th and 24th to complete his *x*-ray treatment (550 "R" units). He has been heard from at frequent intervals since and is in perfect health, entirely free from symptoms.

Discussion.—This patient presents one of the more common errors in diagnosis—the diagnosis of a tumor of the cauda equina as a sciatica. All cases of bilateral sciatic involvement should have a lumbar puncture and manometric determinations to detect evidence of a block of the cerebrospinal fluid. Sciatica is rarely bilateral and all such cases should be considered as due to tumor until proved otherwise. The aggravation of the pain by coughing, sneezing, straining at stool, etc., is almost pathognomonic of a neoplasm of the cauda equina, and when in addition there is present difficulty with bowel and bladder there can be little question of the diagnosis. The specimen removed at operation was lost in the pathologic department, but the tu-

mor was doubtless of the type known as giant-celled tumor of the cauda.

CHONDROMA OF INTERVERTEBRAL DISK

This type of lesion has only been recognized during the past two years, and as yet there is no means by which it can be recognized before operation.

Case VII. Syndrome of Involvement of the Cauda Equina. Partial Cerebrospinal Fluid Block. Laminectomy. Removal of Chondroma. Recovery.—H. S., a Jewish junk dealer, forty-seven years of age was referred to this clinic by Drs. D. B. Rotman and Peter Bassoe of Chicago.

History.—He was admitted on July 3, 1929, complaining of difficulty in walking and pain in the back and down the posterior aspects of both legs. He stated that three or four years ago he had a severe pain in the lower back and consulted a physician who made a diagnosis of sacro-iliac strain, and advised the patient to wear a sacro-iliac belt. He did this and the pain subsided in about a week. During the following years he had similar attacks, about six times a year. With each attack he would again wear the belt and the pain always subsided in two or three days. He was entirely free from pain during the winter of 1928–1929 until on April 25, 1929, when it again recurred. The following day it was more severe and for the first time was associated with pains down the posterior aspects of both lower extremities. On April 27th, while lifting a heavy piece of iron, he suddenly suffered from a very severe pain in the back and legs which caused him to fall. He was unable to move on account of the pain and was carried home. The following week he was hardly able to move because of the severe pain. Two weeks prior to his admission to this clinic he entered the Presbyterian Hospital, Chicago. While there three lumbar punctures were performed. These greatly aggravated his symptoms.

Examination.—The general physical examination was essentially negative. There were no pathologic findings in the upper extremities. Sensory examination revealed a definite hypo-

esthesia to painful and tactile stimuli in the lower extremities; however, no definite upper level could be determined. No alteration of response to thermal stimuli could be detected. The patient's station was normal. His gait was very slow and he was obviously holding his lower extremities very tense to protect himself from the extreme pain which motion elicited. There was a definite weakness of the flexors of the left knee, all other muscle groups were strong. Percussion of the lumbar spine elicited coarse myoclonic twitchings of the muscles of the posterior aspect of the right thigh and of the right calf. There was mild tenderness to pressure over the fourth lumbar spine. Lasègue's sign was positive on the left. The sciatic nerves were not tender to pressure. The abdominal and cremasteric reflexes were distinctly hyperactive as were the patellar and Achilles' tendon reflexes, the right being slightly more marked than the left. Babinski's sign was absent.

A lumbar puncture was performed. It was impossible to obtain any spinal fluid at the third to fourth interspace. (This was also the experience of Drs. Rotman and Bassoe.) The needle was therefore inserted between the fourth and fifth vertebrae. The initial pressure was 120 mm. of fluid. On jugular compression it rose very slowly to 300 mm. and when the compression was released it fell slowly to 180 mm., thus indicating a partial block of the cerebrospinal fluid pathway. About 3 cc. of clear colorless fluid was obtained and $1\frac{1}{2}$ cc. of lipiodol was injected. The patient then was examined under the fluoroscope on the tilting table. The lipiodol descended normally into the sacral sac, but with the patient's position reversed, his head down, it would not pass beyond the fourth lumbar vertebra.

Laboratory examination of the cerebrospinal fluid revealed 198.5 mg. of protein per 100 cc. of fluid. The Wassermann tests on both blood and fluid were negative.

Operation.—On July 6, 1929 a laminectomy was performed. At first the laminae of only the fourth and fifth lumbar vertebrae were removed. At this level the roots of the cauda equina were markedly inflamed and there was no evidence of pulsation. The laminae of the second and third lumbar vertebrae were

then removed, and on incising the dura mater at this level the caudal roots at the level of the junction of the third and fourth lumbar vertebrae herniated out of the dural incision. Above this point the roots were normal in appearance and there was normal pulsation of the cerebrospinal fluid. The roots were then dislocated backward revealing a smooth elevation of the dura mater which lay to the left of the midline. The dura mater was incised over the elevation revealing a yellowish, rub-

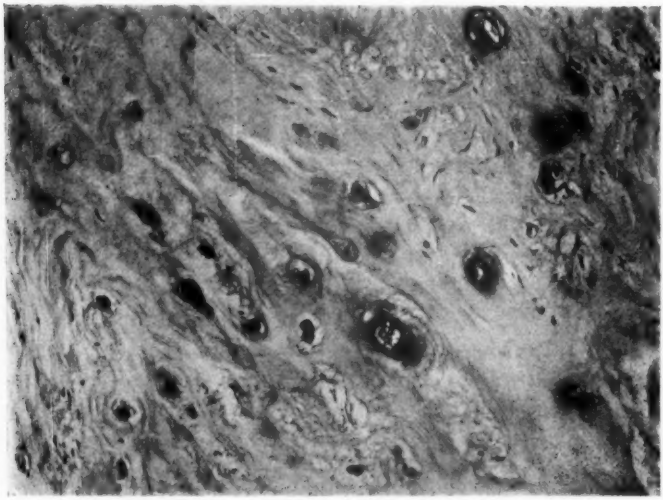

Fig. 110.—Case VII. Chondroma of the intervertebral disk. The irregularity of the cells and the multinucleated and vacuolated cells are typical of a chondroma. Hematoxylin and eosin (× 100).

bery tumor about 1 cm. in length and projecting backward about ½ cm. into the spinal canal. This tumor was firmly attached to the intervertebral disk lying between the third and fourth lumbar vertebrae. It was removed in pieces with a curet.

Postoperative Course.—Following the operation it was necessary to catheterize the patient for three days. Otherwise he made a rapid and uneventful recovery. When discharged on July 24th, eighteen days after the operation, his gait was greatly

improved; he was able to stand on either foot alone, which was impossible before operation and, most important of all, he was free from pain both while quiet and in motion. Sensation was normal. The superficial reflexes were still somewhat hyperactive.

He was last seen on October 15th. At that time his station and gait were perfectly normal, and he had no complaints except for an occasional slight pain in the back on overexertion.

Pathology.—Histologic examination of the specimen removed at operation reveals a typical chondroma composed of fibrocartilage (Fig. 110). There are marked variations in the histologic picture in different areas. Everywhere the cells are irregularly arranged. They vary greatly in size. There are many spindle and stellate-shaped cells of obviously fibroblastic nature. These are scattered irregularly throughout the specimen. The cartilage cells also vary greatly in size and number. Many are small mononuclear cells with a variable amount of cytoplasm. Others are large mononuclear or multinuclear cells with a large amount of cytoplasm containing many vacuoles, the physaliden of Virchow. The proportion of cells to matrix also varies greatly. In some regions we find a large amount of matrix and very few cells, elsewhere the cells, especially of the fibroblastic type, are closely crowded together.

Discussion.—This lesion is probably not infrequent. Some 18 cases have been reported since Stookey first called attention to the condition two years ago.

Surgery of the spinal cord is much more gratifying than surgery of the brain, for tumors of the spinal cord proper are rare whereas tumors of the brain substance are unfortunately more common than extracerebral tumors. The results in these cases give evidence of the extraordinary power of the spinal cord to recover its functions after compression, so that no case should be considered hopeless.

CLINIC OF DR. CARL B. DAVIS[1]

PRESBYTERIAN HOSPITAL

PAUCHET CLOSURE

THE next two patients are examples of the Pauchet method of closure of a colostomy. Both patients had carcinoma of the sigmoid at the level at which the sigmoid is most freely movable. Neither patient showed lymphatic involvement except for a few glands close to the intestine. Both patients were of an age where prolonged anesthesia was not advisable.

The first patient came in with an obstruction that was practically complete. A narrow, annular tumor was found in the sigmoid with one or two glands involved, and they were found close to the gut. There was a long mesosigmoid which made it easy to pull the tumor and approximately 12 inches of gut through the midline incision. Enough gut and mesosigmoid were brought through so that apparently all the involved glands were delivered. The gut was rotated in such a manner that the sigmoid vessels were out of the line of coaptation of the loops of gut. An ileostomy was done at the same time. Twenty-four hours after operation the ileostomy had taken care of practically all the abdominal distention. Twenty-four hours after operation a tube was inserted in the sigmoidal loop proximal to the tumor. At the end of ten days the protruding sigmoid and tumor were removed. Three weeks later the bowel was closed by the Pauchet method.

I am taking the liberty of explaining this method in some detail as I have not seen it recorded in the American literature. It has been published in the Proceedings of the Society of Sur-

[1] Portion of the clinic given before the Clinical Congress of the American College of Surgeons, October, 1929.

gery of Paris, but as this does not have a large circulation in this country it seems fair to repeat it. I saw Pauchet use it in Paris for a primary closure of the large intestine. After resecting a large bowel tumor he uses this method intraperitoneally or extraperitoneally. It seems like an ideal method of extraperitoneal

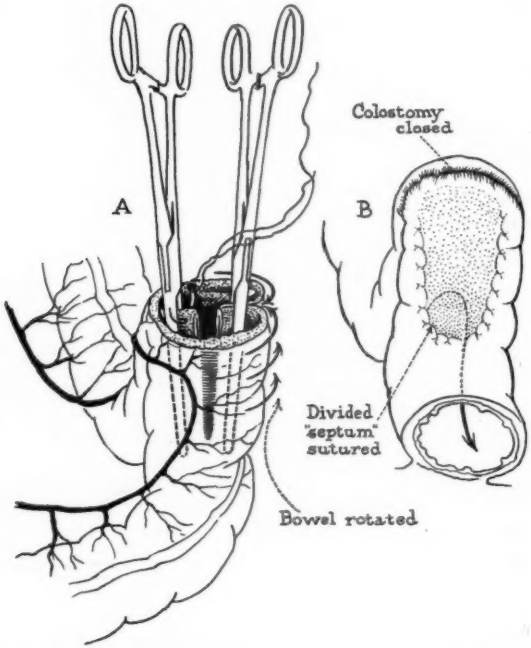

Fig. 111.—A, The blood supply is shown well out of the line of clamps. The suture is starting down the septum which has been severed. B, The suture line in the septum is shown as though the efferent loop were transparent.

closure. Two clamps are slipped over the spur of the colostomy down to whatever depth the surgeon desires. The afferent and efferent loops of gut should be sutured together at the primary operation to eliminate the possibility of intervention of the sigmoidal vessels. The spur is cut down between the clamps and two rows of linen suture are carried down, across, and up the

incised area to approximate the layers of intestine. The colostomy, or that portion of the gut which is protruding through the skin, is mobilized down to the peritoneum, but the peritoneum is not opened. The gut is then turned in in the usual extraperitoneal manner. On the face of it this procedure is so much better than the clamp method that it had a great appeal to me. I have used it eight times in the last two years and have yet to find any weakness in the mtehod. The illustrations are practically self-explanatory.

The next patient was a young woman who entered the Presbyterian Hospital with a record of injury of the duodenum during a gallbladder procedure. On entrance she had the usual digested area of skin about 5 inches in diameter. In the central portion of this area was a crater-like ulcer into the depth of which poured bile-stained fluid and particles of food. She was given methylene blue solution by mouth and in a few minutes the fluid was identified in the crater, indicating the defect in the bowel was not far from the stomach. Having found pancreatic extract in the usual tests in these cases, we assumed without any further investigation that the severe erosion was due to the pancreatic secretion. We know that the acid stomach content passing over the duodenum produces a reflex action resulting in the extravasation of the pancreatic juice. In a number of previous cases we have found it possible to check, to a large degree, the erosion by keeping the stomach content alkalinized as it goes into the duodenum. This patient was given 10 grains of sodium bicarbonate every hour day and night. In forty-eight hours the improvement was astonishing. Repeated efforts were made to plug the defect with barium. The hole in the duodenum was either in such a position or so large that we were unable to make any sort of a plug that would hold. We finally used a modified Beck paste with the melting point well above that of body temperature. A catheter attached to a syringe was carried to the depth of the wound and modified Beck's paste was injected into the tract. The catheter was then slowly withdrawn. A moderate amount of paste in this way was forced into the irregularities of the wound in such a manner

that a portion of it at any rate did not slip into the gut. The wound was then collapsed as much as possible with adhesive tape, and a pyramidal-like gauze pack with zinc oxide was then forced into the crater with rather firm pressure. In forty-eight hours healing was evident, and in two weeks the large defect had pinched down until it was difficult to insert the catheter. The fistulous tract was reinjected with Beck's paste, but the paste constantly disappeared into the intestines. The fistula was then swabbed out with $\frac{1}{2}$ per cent, 2 per cent, and 5 per cent silver nitrate.

At the present time you see the patient still has a very small opening on the abdomen which will not admit the ordinary grooved director. A small silver probe can be passed along the tract. The spot on the dressing which has been present on the patient for three and one half hours, you see, is a very small spot. The patient states that many days there is no stain on the dressing. It is now several months since the first effort at closure was attempted. We have given up the barium injection as there has been a tendency for the barium to collect in the large gut, giving the patient considerable distress. *x*-Ray pictures have shown it at intervals in considerable quantities.

A few months ago in handling an accidental injury of the small gut close to the stomach, we had to contend with a duodenal fistula and ulcer that had a crater for a time approximately 6 inches in diameter. Into this crater came bile, milk, and food particles almost immediately after taking food by mouth. The patient was given sodium bicarbonate, 10 grains every hour day and night. After a week's test with the alkali considerable progress was made in healing. It was explained to the patient that valuable information could be obtained by a withdrawal of the alkaline treatment and the ingestion of acid fruits, and with her consent a series of experiments were attempted to determine what would happen. The patient had been kept on a diet that was entirely free of acids. The diet had consisted largely of milk and an occasional soft egg, with small amounts of cracker or toast. The alkaline treatment was withdrawn

and the same diet persisted in. In twenty-four hours the wound became red and angry and the granulations disappeared like snow in the hot sun. The pain, which is always present in these cases when unprotected by alkalies, returned almost at once. The patient was then put back on alkalies until control had been obtained. Grapefruit and orange juice were then given to the patient with the addition of alkalies. The reaction was almost the same as that with the stomach contents unprotected with alkalies. The granulations again disappeared, the wound became red and angry and the pain returned. The alkalies were then returned to the management and after two weeks a moderate amount of fruit juice was used again as the patient had had so much alkalies it seemed fair to yield to her desire for fruit. The result this time was almost identical with that of the early experimental effort. The granulations began to disappear, the pain returned, and the wound began to break down. From then on for the next month the patient was kept on alkali and acid-free food.

In this patient the entire closure was obtained in six weeks.

Another patient, in whom we had the opportunity of employing the alkaline method, was seen forty-eight hours after operation. Alkaline management was instituted at once and in her case closure of the duodenal fistula was obtained in about a month.

We have used this method in 10 cases. We cannot recommend its trial too highly in injuries similar to those which we have described.

This young woman whom we have shown this morning has been a failure so far as complete closure is concerned, but in her case the fistulous tract was several months old before we started management. In her case I think the fistulous tract has become lined with columnar cells and it will only be when these cells are destroyed that closure will be obtained. In 1 case of this type I was able to recover columnar cells by curetting the fistula. In this woman the fistulous tract had been present for several months. It seems reasonable to suppose that the earlier a case has been put under management the

less chance there will be of columnar cells extending out of the gut and distributing along the diseased area. We know that bladder fistulae close following prostatectomy if the wound is permitted to collapse at an early stage, but in those cases where the fistula has been present too long it is necessary to dissect out the tract before closure is brought about.

INJURY TO THE ABDOMEN WITH AN EXPLOSION OF A VISCUS

THE next patient is a young man who returns today merely for check-over. Approximately a year ago this patient suffered an accident which resulted in an injury of a type which I have never seen before. A few hours after eating a full meal he was caught between a moving crane and the cross-beam of a ceiling in such a manner that an enormous force was exerted first on the left side of the abdomen and gradually shifting toward the right side. My impression is that the stomach and retroperitoneal portion of the duodenum were pressed against the vertebral column. A continuation of this left to right pressure resulted in a forcing of the contents of the stomach into the duodenum and also preventing the contents from passing out of the retroperitoneal portion of the duodenum. There was an overdistention of the gut until rupture occurred.

The outcries of the patient resulted in a checking of the action of the machinery just before he was crushed.

The patient showed some shock, extreme weakness, and a moderate amount of pain. He was transferred to the hospital at once and within two hours after the injury was in the operating room.

He showed a normal temperature, pulse of 100, leukocyte count of 17,000, and a very slight rigidity of the upper abdomen. There was a small amount of tenderness in the upper right quadrant.

A rupture of an abdominal organ was suspected and the abdomen was opened at once. On entering the peritoneal cavity no blood or free fluid was seen, ruling out a severe rupture of any of the parenchymatous organs. A rapid survey of the stomach and the visible portion of the duodenum showed no leak. The entire intestinal tract was then gone over inch by inch, looking for a possible rupture.

With negative findings in the lower abdomen and because of the man's record of pain in the upper abdomen a further search was made of the upper area. Approximately 1 inch median to the right kidney a small discoloration was seen beneath the peritoneum. This area was broken into and a small amount of bile-stained fluid and blood escaped. The area was enlarged and the examining finger was passed into a defect on the posterior wall of the junction of the second and third portions of the duodenum. The third portion of the duodenum was mobilized, turned up and the defect in the musculature was closed by interrupted linen sutures. The tip of the omentum was then carried up into the retroperitoneal area in the region of the great vessels and pancreas. The omentum was stitched over the extraperitoneal portion of the gut in such a manner as to reenforce the line of suture. As far as possible the duodenum was stitched back into normal position. A cigaret drain was carried down to the peritoneal defect. The drain was removed on the fourth day. The patient made an uneventful recovery and had practically no distress.

This case is presented today as showing one of the most unusual types of explosion of an abdominal viscus through force applied upon the surface of the abdomen without cutaneous injury. It is interesting because of the fact that the area injured showed so few signs that it was overlooked on first examination. It is interesting because of the problem that it presented in making a peritoneal closure in an extraperitoneal area.

The patient has no discomfort or symptom referable to the area injured.

CLINIC OF DRS. DAVID C. STRAUS AND
HENRY H. RUBIN[1]

MICHAEL REESE HOSPITAL

ANALYSIS OF 100 CONSECUTIVE THYROIDECTOMIES FOR GOITER WITH HYPERTHYROIDISM

THE data that I wish to present are an analysis of 100 consecutive thyroidectomies performed at the Michael Reese Hospital by one of us (D. C. S.). The majority of the cases were on the thyroid group of the Michael Reese Hospital and credit is due and hereby gratefully acknowledged to Drs. Walter W. Hamburger and Solomon Strouse, internists on the group, Dr. Walter S. Priest, who is in charge of the electrocardiographic work, Miss Barrens, of the Social Service Department, and Drs. Graef, Lev, and Rubin for the follow-up work in the Outpatient Department.

No patient coming to the hospital with hyperthyroidism during this period was refused operation and the series presents an unusually toxic group of cases. No case was considered one of hyperthyroidism in which the basal rate was under +15. Of the 100 cases, 4 showed a basal rate of between 90 and 100; 2, between 80 and 90; 13, between 70 and 80; 14, between 60 and 70; 16, between 50 and 60; 17, between 40 and 50, and 34, between 15 and 50, of which more than one third were +30. Forty-nine, or almost exactly one half, were over +50; 66, or two thirds, were over +40, and 34, or one third, were under +40 (Table 1 on page 268).

[1] Presented before the American College of Surgeons at Michael Reese Hospital during the Clinical Congress, October 14–18, 1929. From the Goiter Group of Michael Reese Hospital.

TABLE 1

BASAL METABOLIC RATE

90–100	4	99.7	90.8	97.9	91.0						
80– 90	2	81.3	83.8								
70– 80	13	74.3	79.8	72.0	75.5	78.9	74.0	70.9	72.6	74.3	74.3
		74.9	77.0	75.3							
60– 70	14	67.9	65.4	68.1	65.6	61.9	66.5	62.9	62.8	61.5	64.5
		61.8	63.7	62.6	65.9						
50– 60	16	50.4	54.7	55.2	55.9	59.0	57.4	51.1	59.7	58.0	50.9
		55.4	58.4	59.3	50.0	55.0	59.4				
40– 50	17	49.3	45.6	48.0	42.3	43.0	49.1	41.8	4.26	47.6	48.8
		45.8	49.0	44.0	45.7	40.2	47.0	47.5			
15– 40	34	36.0	31.1	38.4	31.8	35.7	34.6	39.8	36.0	39.8	35.0
		32.7	37.0								

(More than ⅓ over 30.)

Almost exactly
49 or ½ over 50
66 or ⅔ over 40
34 or ⅓ under 40 (of which 12, or more than ⅓ were over 30.)

Preoperative Study and Preparation.—No effort was made to differentiate between cases of exophthalmic goiter and cases of so-called toxic adenoma. As I pointed out in a recent article,[1] viewed from the clinical evidence and the response to iodine therapy, it seems more than likely that exophthalmic goiter and toxic adenoma are only variations of a single morbid state. Exophthalmic goiter may be looked upon as the more acute type, and toxic adenoma as the more chronic, slowly progressing type. In my experience both types respond about equally well to iodine, and iodine was given preoperatively in all the cases of this series.

On entrance to the hospital each patient had a basal metabolic rate taken. Lugol's solution was then begun. After iodine had been administered for one week, a second metabolic rate was taken and repeated every three days thereafter up to the time of operation. The dosage of Lugol's solution varied from time to time in different individuals, but the usual dosage was 10 minims three times a day. There was no attempt made to give iodine for any definite period of time, the duration of administration depending on the results obtained. An attempt was made to have the basal metabolic reading come down to below +30 or, if possible, to below +20. In the majority of the

[1] Straus, David C., "Present Day Views on Goiter, from the Standpoint of the Surgeon," Illinois Med. Jour., Aug., 1928.

cases Lugol's solution was administered for as long as two to three weeks and in a few cases even longer. In those cases in which the basal metabolic rate is in the 20's, it might have been possible with continued administration, to accomplish even further reduction, though this was not deemed essential. Where the administration had been continued as long as from two to three weeks and the basal metabolic rate was in the 20's, it was considered best to operate with a basal rate that was falling rather than to wait until there would be no further reduction or even a possible rise.

Analysis of the Cases.—In order to analyze best just what was accomplished in these 100 cases, they were grouped according to the last preoperative basal metabolic rate. There were 10 cases in which the basal metabolic rate at the time of operation was from +20 to +25. The average basal metabolic rate of these cases on admission was +48.8. The average basal metabolic rate at the time of discharge from the hospital was +9.3 (Table 2 on pages 270 and 271).

There were 9 cases in which the basal metabolic rate at the time of operation was between +25 and +30. The average rate of these cases on entrance was +49.6. At the time of discharge the average rate was +3.9.

There were 8 cases in which the basal metabolic rate at the time of operation was from +30 to +35. The average rate on entrance was +50. On discharge from the hospital the average rate was +3.1.

There were 8 cases in which the basal metabolic rate at the time of operation was from +35 to +40. The average rate of these cases on admission was +63.8. On discharge the average rate was +6.8.

There were 9 cases with a basal metabolic rate at the time of operation of +40 to +45. The average rate on entrance was +59.9 and on discharge +3.9.

There were 5 cases in which the basal metabolic rate at the time of operation was +45 to +50. The average rate on entrance was +70.5. On discharge from the hospital the average rate was +10.8.

TABLE 2

BASAL METABOLIC RATES AT TIME OF OPERATION BETWEEN

		+20–25			+25–30			
No.	Case.	Entr.	Op.	P. O.	No.	Case.	Entr.	Op.	P. O.
1	17	+54.7	24.7		1	15	+50.4	27.4	+ 5.8
2	21	+38.4	20.3	+18.6	2	60	+58.4	28.4	− 7.5
3	24	+55.9	22.4		3	64	+47.6	27.9	+25.0
4	42	+51.1	24.8	+17.2	4	65	+59.3	28.0	+12.3
5	46	+15.6	21.2	+22.5 (Ca)	5	66	+74.9	26.2	+ 4.6
6	63	+42.6	20.3	+16.6	6	68	+48.8	27.9	+ 1.4
7	72	+36.0	24.7	− 1.4	7	71	+23.1	29.8	+ 6.8
8	75	+32.7	23.1	+ 3.7	8	74	+35.0	29.8	−11.0
9	77	+91.0	20.1	+ 7.1	9	87	+49.0	27.5	− 2.0
10	84	+45.8	23.3	+ 8.2					
11	86	+50.0	21.5	+ 4.4	Average		+49.6		+ 3.9
Average		+45.8		+10.7					
Corrected (Omiting Ca)		+48.8		+ 9.3					

		+30–35			+35–40			
No.	Case.	Entr.	Op.	P. O.	No.	Case.	Entr.	Op.	P. O.
1	18	+31.1	+33.8	+13.1	1	5	+74.3	37.0	
2	10	+90.8	30.1	+ 9.3	2	32	+61.9	37.9	
3	39	+57.4	32.0	+ 5.1	3	36	+78.9	38.6	− 7.0
4	43	+34.6	30.8	− 5.9	4	49	+72.6	37.1	+ 4.6
5	45	+59.7	32.6	+ 0.5	5	73	+39.8	39.8	+16.1
6	70	+26.3	34.5	− 7.0	6	85	+81.6	36.5	− 6.1
7	80	+63.7	32.6	+ 9.9	7	91	+55.0	35.0	+26.5
8	82	+37.0	34.5	+10.2	8	94	+47.0	37.8	
Average		+50.0		+ 3.1			+63.8		+ 6.8

		+40–45			+45–50			
No.	Case.	Entr.	Op.	P. O.	No.	Case.	Entr.	Op.	P. O.
1	20	+45.6	44.8		1	31	+65.6	48.0	+24.4
2	22	+55.2	44.9	− 0.4	2	34	+66.5	45.3	+18.0
3	25	+59.0	43.0		3	40	+74.0	46.4	+10.4
4	44	+70.9	43.8	+ 5.7	4	83	+62.6	45.5	+ 0.5
5	52	+49.1	40.8	+ 8.6	5	97	+83.8	46.9	+ 0.7
6	54	+50.9	43.0		Average		+70.5		+10.8
7	62	+74.3	44.1	− 4.2					
8	92	+59.4	43.0						
9	100	+75.3	40.0	+ 9.9					
Average		+59.9		+ 3.9					

		+50–55			+55–60			
No.	Case.	Entr.	Op.	P. O.	No.	Case.	Entr.	Op.	P. O.
1	67	+64.5	54.0	+39.4	1	27	+68.1	57.2	
2	98	+77.0	53.1	+ 9.5	2	35	+43.0	56.1	+24.4
Average		+70.7		+24.4	3	47	+58.0	56.8	+50.9 (Ca)

+60–65			+65–70		
None	1	13	+67.9	65.0	+24.6
70–75			+75–80		
None	1	14	+97.9	77.7	Died
			+80–85		
	1	26	+65.4	+83.0	+10.0 Pregnancy

In the cases in which the basal metabolic reading at the time of operation was over +50, the number was so small that the details can best be appreciated by seeing Table 2. There was only 1 case in which the basal metabolic rate at the time of operation was over +80. This patient came into the hospital in the early months of pregnancy, with a basal metabolic rate of +65.4. In spite of the administration of Lugol's solution her basal metabolic rate steadily increased. Operation seemed out of the question if it was at all possible to lower the rate by any nonoperative means. Consequently, an attempt was made to try *x*-ray treatments locally. Four treatments were given in three weeks, but in spite of this her basal metabolic rate continued to go up and it reached +83 when operation seemed imperative. Polar ligation followed by a right lobectomy, and eighteen days later a left lobectomy was done. The postoperative reading on discharge from the hospital was +10, and the patient went on to term.

Cases with Auricular Fibrillation (Table 3 on page 272).—There were 8 cases in the series presenting auricular fibrillation at the time of operation. These patients showed an average metabolic rate at this time of +41.5 and an average pulse rate of 92.7. Operation in these cases was even more radical than usual and 1 of these cases (Case 83) was the only one in the 100 that was followed by tetany.

Results.—*Insufficient Operation.*—Only 1 case in the 100 (Case 13) returned with symptoms of hyperthyroidism, due to the removal of an insufficient amount of the gland. This patient had a basal metabolic rate on entrance of +67.9. After the usual preoperative preparation the basal rate was only reduced

TABLE 3

CASES WITH AURICULAR FIBRILLATION

No.	Case.	Pulse at Operation.	B. M. R at Op.	B. M. R. after—	On discharge.	Result.
			+74.3			
1	62	80	+44.0	− 4.2	Auricular flutter.	Sinus arrythmia.
2	73	112	+39.8	+16.0		
			+62.8			
3	83	102	+45.5	+ 0.5	P. 80 (tetany).	
			+59.4			
4	92	92	+43.0	...	P. 72.	
			+65.9			
5	96	96	+12.9	+ 2.0	P. 72.	Still fibrillating.
		109	+77.0			
6	98	84	+53.1	+ 9.5	.....	Still present.
		92	+28.7			
7	99	76	+16.4	+12.0	P. 76.	
		120	+97.8			
8	14	100	+77.7	...	.....	Died.

to +65. She was one of the few cases in which multiple stage operation was performed; polar ligation, later resection of one lobe, and finally resection of the remaining lobe. On discharge from the hospital her basal rate was +24.6. She was later given *x*-ray treatments with a final reduction of her basal metabolic rate to +8.7 and complete freedom from symptoms.

Recurrence.—Only one patient in the series had a true recurrence (Case 10). She was admitted with a basal metabolic rate of +90.8. After administration of 15 minims of Lugol's solution three times a day for nine days, the basal metabolic rate was reduced to +30.1. After a subtotal thyroidectomy in one stage she was discharged with a basal metabolic rate of +9.3. She remained well for two and one half years, at which time after an attack of acute tonsillitis, she returned with marked symptoms of hyperthyroidism and a basal rate of +41.4. This is the only patient in the series in which reoperation was necessary. A subtotal thyroidectomy was done in one stage and the patient left the hospital with a basal metabolic rate of −12. This second operation was extremely radical and this patient was one of four in which the operation was complicated by paralysis of one vocal cord.

Complications.—Paralysis of one vocal cord occurred in 4 cases (Cases 10, 17, 84, 98).

Only 1 case in the 100 showed obvious clinical evidence of tetany (Case 83). No other cases of tetany were observed.

Deaths.—There were three deaths in the series, though one of these cannot properly be considered as an operative death. This patient (Case 14) was admitted to my service with auricular fibrillation, a pulse of 120, and a basal metabolic rate of +97.8.

Fig. 111*a*.—Photograph of patient (Case XIV) whose death following operation was believed to be due to the administration (without my knowledge or consent) of two doses of scopolamine-morphine. The patient had been treated medically, with administration of Lugol's solution for one year, before he came under my care, was fibrillating and is a striking example of the dangers of long continued medical care with long continued administration of iodine in a toxic patient. Operation consisted of removal of one lobe, was easily and quickly performed, as preliminary polar ligation, both right and left, had been performed weeks before. He died one hour after operation from cardiac failure, which was believed was probably due to the administration of the scopolamine-morphine.

He had been treated medically for almost two years with high caloric diet, rest in bed, and iodine for many months. He presented the most extreme case of exophthalmos, a huge goiter, and should probably have been refused operation as an impossible risk. However, it seemed that his only chance lay in an operation and it was deemed essential to do this in multiple stages. Polar ligation was first done, but without any benefit and three months later a lobectomy was decided. The morning of this

operation the intern administered two doses of scopolamine and morphine without my knowledge or consent. The operation was quickly and easily performed but the patient died the same day. His death was attributed by me and by others to the scopolamine and morphine, which should certainly be given in extremely guarded doses, if at all, to patients presenting serious cardiac damage. Personally, I avoid scopolamine in all my goiter cases.

There were only two real postoperative deaths. One of these (Case 20) was in a girl, twenty-three years of age, whose metabolic rate on admission was +45. Lugol's solution was administered for twenty-two days and her basal metabolic rate at the time of operation was +44.8. Death occurred three hours after operation, apparently due to tracheal collapse. The other patient (Case 54), a girl fifteen years of age, was admitted with a basal metabolic rate of +50.9. After thirty-seven days on Lugol's solution the rate only came down to +43. At this time both poles were ligated and three and one half weeks later subtotal thyroidectomy was performed. The patient had a typical postoperative thyroid crisis from which she did not recover. This case is instructive in pointing out the small amount of benefit of iodine administration in children and emphasizing greater surgical risks of thyroidectomy for severe hyperthyroidism in children.

100 CONSECUTIVE CASES WITH HYPERTHYROIDISM

(None were refused operation)

UNTOWARD RESULTS

Complications:

Paralysis of one vocal cord ..... 4 (Cases 10, 17, 84, 98)
Tetany ..... 1 (Case 83, a fibrillator)

Deaths:

(*a*) From scopolomine-morphine (1).

Case 14: A case improperly handled with Lugol's solution for one year before coming under my care. Marked cardiac damage and auricular fibrillation.

(*b*) Postoperative (2).

Case 20 (from tracheal collapse)—Twenty-three years old, +45
Lugol's twenty-two days, +44.8
Case 54 (from thyroid crisis)— Fifteen years old, +50.9
Lugol's thirty-seven days, +43

CLINIC OF DR. GOLDER L. McWHORTER

PRESBYTERIAN HOSPITAL

OPERATIVE TREATMENT FOR EXTENSIVE HYPOSPADIAS

THIS patient demonstrates the good results that may be obtained in the surgical correction of an extensive hypospadias.

Case No. 238621, five years of age, was admitted July 12, 1929. He complained of inability to urinate without sitting down, due to marked hypospadias.

At birth the attending physician stated that it was impossible to determine definitely the sex. The external genitals were more like a girl, or a hermaphrodite, than a boy. Fortunately the physician recognized that this was a boy with extensive hypospadias and undescended testicles.

Past History.—Patient has had scarlet fever and pertussis, but rarely a sore throat. He has always had a moderate internal strabismus, and has stuttered some.

There were two older children perfectly well and no history of congenital deformities.

Physical Examination.—The child was well nourished and had the general appearance of a boy. He stuttered some, but was mentally bright. There was a moderate external strabismus, but there was nothing otherwise unusual in his general condition.

Examination of the genitals revealed that the penis was bound down to the perineum. The prepuce was redundant posteriorly, and absent anteriorly. The glans was flattened with no urethral opening, and the frenum was absent. The urethral opening was in the midperineum, a short distance below the bound-down glans. The scrotal sac was cleft, undeveloped, and indicated by folds of skin at the sides of the perineum, resembling a labia majora. No testicles could be palpated. The urethral

opening terminated abruptly in the perineum with no continuation of a mucus-lined groove in the midline.

The first operation was performed July 13, 1929. It consisted of freeing and straightening the penis. A transverse incision was made just below the glans, curving around rather close to the end of the penis (Fig. 112, 1). By cutting deep cicatricial-like bands, the penis could be straightened. This made a large diamond-shaped raw area, which was obliterated by pulling the lateral ends of the skin incision together. This brought the skin edges together in the midline where they were sutured with fine catgut and skin silk (Fig. 112, 2). The penis was now held in the straightened position. The wound healed well.

The second operation in which the reconstruction of the urethra was done in one stage was performed October 29, 1929. A perineal incision was first done going posteriorly to the urethral opening. A small No. 12 F. catheter was passed into the bladder and fastened for temporary drainage. A piece of fine tube was inserted forward for a short distance into the urethra and fastened in the incision (Fig. 112, 7). A transverse incision was made just beneath the edge of the glans, passing completely around the prepuce. This was extended quite deep underneath so that the penis could be slightly overextended, making a small diamond-shaped raw surface beneath. A generous tunnel was cut through the center of this raw surface extending upward through the glans, with a long narrow knife for the later insertion of the new urethral tube. A skin flap was now outlined for the formation of a new urethra in the perineum and penis, by making an incision about $\frac{1}{3}$ inch along either side of the median line (Fig. 112, 3, *c*, *d*). These extended upward and deviated outward about $\frac{1}{3}$ inch, when the diamond-shaped raw area was reached to form a pedunculated skin flap by extending completely around the prepuce parallel to the earlier incision around the glans. This flap of skin around the prepuce was freed except at the two pedicles below, forming a hood or "stole," and drawn forward over the glans (Fig. 112, 4, *a*, *b*). The two edges of these flaps were sutured together with the skin

surface on the inside, forming a tube, using 00 chromic catgut (Fig. 112, 5, *a*, *b*). The distal end was cut open to make the outlet of the urethra. This tubular flap of prepuce was now inserted from below and drawn upward into the newly made tunnel in the glans (Fig. 112, 6). A few silk sutures were inserted to hold the end at the meatus. The lateral edge of the

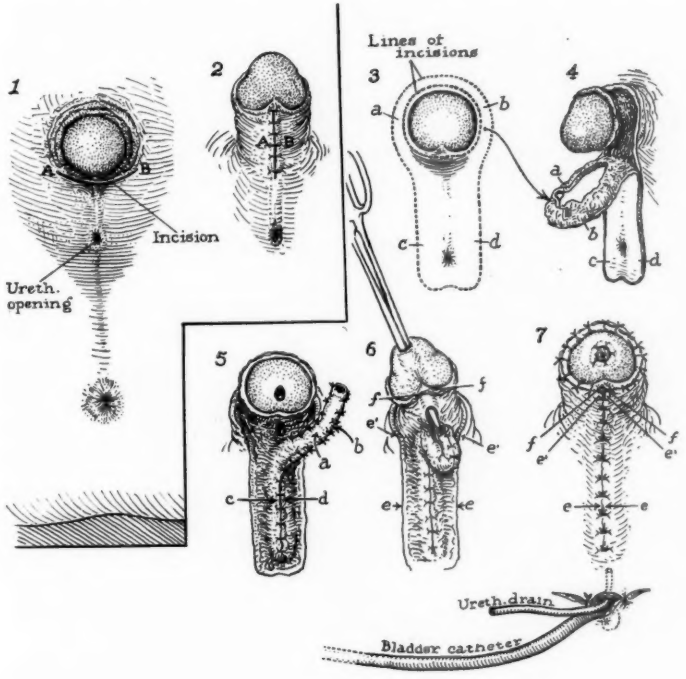

Fig. 112.

skin flaps (*c*, *d*), continuous below with this tube, and previously outlined along the midline, were freed slightly, and the edges sutured together with invagination, forming a complete new urethral channel, extending below, around the former urethral opening, and above through the prepuce tubular flap into the glans (Fig. 112, 6). Continuous and occasional inter-

rupted 00 chromic catgut sutures were used. The raw surface extending below and on either side of the new urethra was obliterated by undercutting the skin edges (*e*, *e*), laterally, and bringing them together in the midline, using interrupted sutures of fine silk. When the glans was reached the defect extending around the prepuce was obliterated by suturing the

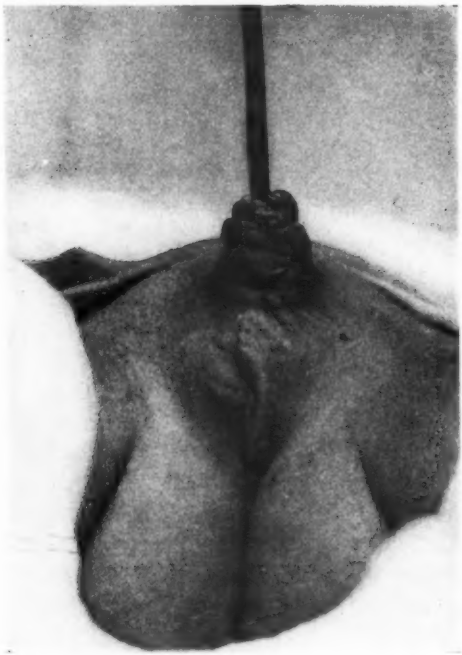

Fig. 113.

edges (e^1, *f*) together with fine chromic catgut, similarly to that done after a circumcision. The penis now stood up very well with about 2 inches of reconstructed urethra (Fig. 112, 7). There was no tension on the skin except a slight amount near the base.

Postoperative Course.—The urine passed through the perineal catheter until about about the fourth day, when it became oc-

cluded, some passing along the urethra and out the newly formed meatus, and some through the perineal incision around the tubes. The tubes were removed and some redness developed around the wound, but this gradually cleared up, leaving a small leak near the base of the penis. The meatus scabbed shut repeatedly, leading to a gradual tendency for the meatus to heal shut except for a small opening.

Thirteen days after operation, the meatus was slit open again where it had grown together for about 1 mm. at the dorsal four fifths of the opening. The urethra beneath this part was freed slightly and sutured again to the edge of the glans. There had been no contraction of the urethral tube, merely a retraction. The entire new urethra now permitted the passage of a No. 12 F. catheter.

Nine days later a small cuff of skin was dissected up about the small leak in the urethra at the base of the penis, and the edges invaginated. The skin was undercut at the sides and sutured together in the midline. The wound in the perineum which was made for temporary bladder drainage had completely healed, and the urine passed out the meatus. Sounds were again passed so that a No. 12 F. catheter could be inserted through the new urethra. These will be passed at intervals in the future, since there is always a tendency for skin flaps used for this purpose to contract.

Report four months after operation: The boy was standing up urinating with no difficulty and had started to school. The result was very satisfactory.

Discussion.—Hypospadias is a congenital absence or lack of development, usually of the distal portion of the urethra, with an opening at some point along its course. It is the result of failure of normal fusion of the genital folds. The upper wall of the urethral canal frequently persists as a shallow groove on the under surface of the penis or there may be a fine scarlike cord indicating its location. The glans is usually broadened and bent downward, and held in this position by a cicatrix-like tissue which may be the undeveloped corpora cavernosa urethra. The scrotum is frequently cleft and when the testicles are unde-

scended the scrotal tissues resemble the labia majora. At birth, such a condition is difficult to differentiate from the female genitals or the rare hermaphroditism. However, the presence of a vagina and a rectal examination may help in the determination of the sex.

Three varieties of hypospadias may be described. First, the balanic or glandular, in which the urethral orifice opens just behind the margin of the glans penis. Second, the penile, in which the urethral orifice opens at some point along the body of the penis in front of the scrotum. Third, the perineal, in which the urethral opening is behind the scrotum, if developed, and near the central point of the perineum.

The preferable age for operation is from four to six years. In the perineal type it should be completed before school age. Later stages may be done satisfactorily up to eight or ten years, after which tearing out of the stitches may occur from postoperative erection.

The balanic type occurs frequently. The frenum and under surface of the prepuce are absent and the glans is flattened with its anterior portion usually bent and held downward. It is usually unnecessary to do anything for this deformity. The Beck-Hacker operation of freeing the end of the urethra and pulling it through a new opening in the glans frequently leads to retraction and scarring.

The penile type of hypospadias is also rather common. This, at first, often appears to be the balanic type, due to the bending downward of the glans. After straightening the penis, a considerable defect is seen to be present in the penile urethra. In the repair of a short defect in the distal end of the urethra, the operation as described by Dr. Bevan[1] is very satisfactory. He dissects up a large area of skin about the external orifice in the form of an umbrella. This forms a funnel-like extension to the urethra, and is pulled through a new opening in the glans where it is attached.

The perineal type of hypospadias is uncommon and frequently associated with undescent of the testicles and a cleft scrotum. Here it is necessary to sit down in order to urinate.

The methods used for its correction have been numerous, none of them being consistently followed by good results. The first step in any of them is to straighten the penis. The reconstruction of the urethra may then be accomplished in either one or two stages, as desired. The reconstruction operation in one stage developed and modified by Thompson[2] from the methods, not entirely satisfactory, of Russell and Duplay[3] have been used in this patient with only slight changes (Fig. 112). Russell cut off any redundant portion of the prepucial tube projecting beyond the meatus. This should rarely be done, since there is considerable shrinkage. He also cautions against including any of the perineal skin in the new urethra, using only the mucosa-covered, partially developed urethral groove. This groove is not always present or sufficient to use in the formation of a good-sized urethra. Consequently, any available skin must be used since it is necessary to have a good-sized urethral channel.

Breaking down of the wound will frequently result if urine is permitted through the new urethra, resulting in failure and increasing difficulty in any future plastic.

Thompson stated that a small rubber drain or a few strands of silkworm-gut should be inserted into the urethra anterior to the perineal bladder drain, since there is an uncontrollable tendency, after a few days, for the urine to pass along the urethra. Some prefer an abdominal temporary bladder drainage. Beck[4] maintained that a temporary bladder drainage was unnecessary if the skin edges had been carefully united, and he recommended fine horsehair throughout. The objections to temporary diversion of the urine are not equal to the advantages of this procedure, especially in the one-stage operation.

The reconstruction may be done in two stages, first tubulizing the glans and later turning in the flaps about the remaining defect in the urethra. Where the congenital opening is located so far posteriorly that a perineal drainage of the bladder is impossible, a suprapubic cystotomy should be done at the second stage. Rarely will an indwelling catheter be satisfactory at this time.

Another method of reconstructing the distal urethra from the

prepuce is utilized by C. H. Mayo.[5] After the penis has been straightened and the wound consolidated, a long flap is cut from the dorsal surface of the penis with its base near the margin of the prepuce. This is freed, except at its base, and the edges sutured together with the skin surface on the inside. A tunnel is made through the glans, reaching a point near the urethral opening and the newly formed tube is pulled down through it and sutured in this position. The raw surface on the back of the penis is obliterated by bringing the skin edges together. After healing the base of the flap is cut from its attachments to the prepuce. At a later operation, the end of this prepucial tube is united to the end of the urethra and followed by a temporary perineal drainage of the bladder. A small urethral fistula, which may complicate any operation, may close spontaneously, but if it persists the edges may be freed and invaginated, bringing the skin over it. A urethral catheter may be of value for a few days.

After practically all reconstruction operations on the urethra there is a tendency for contraction and stenosis, especially at the meatus. This must be prevented by early and repeated passage of sounds, preferably through the entire new portion.

The principles of plastic surgery must be understood and an operative scheme planned out before attempting any operation on a case of hypospadias. The most meticulous care must be given to preservation of the blood supply in the flaps and to accurate suture, followed by careful postoperative care, in order to obtain satisfactory results.

BIBLIOGRAPHY

1. Bevan, A. D.: Hypospadias, Jour. Amer. Med. Assoc., 68, 1032, 1917.
2. Thompson, J. E.: A Study of Modern Operations in Hypospadias from an Anatomical and Functional Viewpoint, Surg., Gyn., and Obstet., vol. 25, 411, 1917.
3. Russell, R. Hamilton: Operation for Severe Hypospadias, Brit. Med. Jour., 2, 1432, November 17, 1900.
4. Beck, C.: Hypospadias and Its Treatment, Surg., Gynec., and Obst., 24, 511, May, 1917.
5. Mayo, C. H.: Hypospadias, Jour. Amer. Med. Assoc., April 27, 1901.

LIGATION OF BOTH THE FEMORAL ARTERY AND VEIN IN THROMBO-ANGIITIS OBLITERANS. REPORT OF THREE CASES

Introduction.—The treatment of a type of gangrene classified as thrombo-angiitis obliterans and occurring in males in the prime of life is a difficult problem. Various authors report a delay or decreased frequency of amputations following many procedures which alone or in combination are of some value.

Recumbency, heat, large quantities of Ringer's solution by the duodenal tube, intravenous administration of salt solution, sodium citrate and sodium iodide, hyperemia, and postural exercises have all been utilized with some good results. Phillips[1] has observed relief following roentgen therapy. Periarterial sympathectomy has given good results which have usually been only temporary.

Allen,[2] in reporting results of bilateral lumbar ganglionectomy, stated that there was not only arterial occlusion but a lack of maximal vasodilatation in thrombo-angiitis obliterans. He stated that removal of the lumbar sympathetic chain is unsuccessful in two types of cases, those in which the organic occlusion rapidly and progressively involves more of the arterial tree and produces death of tissue in spite of utilization of all available blood supply, and those in which further vasodilatation cannot be produced by this procedure. Patients are selected for operation depending upon the vasomotor vasodilatation. Allen stated that only about one out of seven is found to be suitable for lumbar ganglionectomy.

The method evolved by Brown[3] is used for the determination of the available vasodilatation by giving typhoid vaccine intravenously.

Allen and Smithwick[4] found that the vasodilatation in the extremity following intravenous injection of typhoid vaccine was similar to that following sympathectomy. They have observed definite relief from pain and improvement in the local appearance of the lesions after repeated injections, in some of the

cases extending over several months. They believe that it hastens an adequate collateral circulation. Although of some value, it has been difficult to continue the injections in many cases due to the chill and unpleasant reaction.

Allen and Meyerding[5] emphasize the danger of incision of the toes or removal of the toenails, although amputation may be done in selected cases. In those extremities requiring amputation they were able to amputate successfully below the knee in 80 per cent of their cases, but emphasize careful selection and after-care.

The suggestion of Lewis and Reichert[6] of ligating the femoral artery below the profunda for thrombo-angiitis obliterans has stimulated new interest in the surgical treatment of this insidious disease.

Due to the lessened frequency of gangrene in a large series of clinical cases reported by Makins[7] and others by ligating the companion vein where it was necessary to ligate the artery to an extremity following injury and from experimental work of others, I have been led to try ligation of both artery and vein in the treatment of thrombo-angiitis obliterans.

CASE REPORTS

Case I.—J. E., a man aged forty-five and born in Ireland, was first examined January, 1927. His chief complaints were swelling of the right foot and ankle; pain in the right leg and foot; inability to walk on the foot due to pain and swelling; ulceration about medial maleolus of the foot.

Onset and Course.—The disability started about one year ago, 1926, with an acute area of inflammation over the medial side of the ankle. Following hot application, the skin broke down and an ulcer resulted. Since then, the foot and leg have been getting gradually worse. The swelling would increase if he tried to be up. On letting the foot hang down there was constant severe pain. With the leg elevated there was still some pain and swelling. He was unable to put weight on the foot due to the swelling and unable to flex the foot at right angles. This disability has persisted during the previous year.

Past History.—The patient came here from Ireland at the age of twenty-five years. Family history was negative. He has had no previous serious illnesses.

He has been a worker in sewers for twenty-five years. He was right handed. Frequently he would get his feet cold and wet. He wore rubber boots practically all of the time. He never smoked or used tobacco. No previous operations have been performed.

Physical Examination.—January, 1927, the patient was a very tall man with a big frame weighing about 230 pounds and his general condition was excellent. He was well muscled and very strongly built; big chest and body, lower extremities lean and well developed; no varicose veins were evident in either leg. Right leg was edematous, cyanotic, and cold in the lower two thirds. There was considerable pitting on pressure over the lower leg and foot. There was some equinus of the foot and the patient was unable to move the ankle due to the edema. There was an ulcer in the region of medial maleolus about 2 cm. in diameter. No pulsation of the dorsalis pedis artery could be felt but the edema interfered with palpation.

Laboratory examinations showed Wassermann negative, urine and blood examinations normal and blood pressure 135/80.

The first operation was done on January 19, 1927. A periarterial sympathectomy was performed on the femoral artery in Hunter's canal. The artery seemed somewhat smaller than normal. There was occasional patchy calcification observed in the wall. Following this, there was immediate improvement with diminution of the edema. Pulsation of the dorsalis pedis artery could be felt and the foot was warm. The ulcer gradually healed in the next four weeks and the edema entirely disappeared. Following this, the patient was up and walking with no complaints. Gradually, after about six months, during which he was up and walking about every day, there was a return of the edema of the leg and foot which became cold again. Seven months after the sympathectomy, an ulceration started between the first and second toes associated with constant severe pain, and a recurrence of the ulcer over the ankle.

An examination on August 16, 1927 showed that the leg was again cold and cyanotic in the lower two thirds, with pitting on pressure, following two weeks complete rest in bed. There were two ulcers on the foot, one small one below the medial maleolus and one between the first and second toes. There was no pulsation of the dorsalis pedis artery felt.

Second operation was done on August 17, 1927. Ligation of both the right femoral artery and vein in Hunter's canal with heavy silk was done. Following this operation, there was immediate relief of pain in the ulcer between the toes, rapid clearing up of the edema and cyanosis of the leg. There was a sudden change on the surface of the foot and leg from cold to warm. No pulsation of the dorsalis pedis was evident. The ulcers on the toes and foot promptly healed and on September 4, 1927, two and one half weeks after ligation of the femoral artery and vein, there was no edema of the leg and foot, and they were greatly improved in warmth.

Examination on December 28, 1927 showed that there was no complaint. The patient was up and about. The leg swelled some every day, but was moderately warm.

About April 1, 1928 there was recurrence of a smaller ulcer about the medial maleolus but he continued to be on his feet daily thirteen to fourteen hours a day following ligation of the artery and vein. On June 14, 1928, the leg was somewhat edematous and the ulcer was still present. The patient was put to bed in the hospital and the ulcer healed within a month. He was discharged and on his feet again without recurrence of ulcers for about five to six months, when the ulcer returned above the medial malleolus.

Examination on March 10, 1929 revealed that there was a small ulcer over the medial malleolus with some redness about the ankle; less about the foot. Some edema of the leg with pitting was present. No pains were complained of since ligation of the femoral artery and vein.

Vaccine treatment: On March 10, 1929 he was given mixed typhoid and paratyphoid vaccine intravenously. This was repeated every two weeks for three times, each one followed by a violent chill and fever of 104 F. There was improvement in

the foot a few days after each injection with less edema and increased warmth. The patient objected to further vaccine due to the violent chill. The foot and ulcer improved somewhat for a couple of months and then became worse in June, 1929, when the patient had hospital rest for a couple of weeks with healing of the ulcer. The ulcer remained healed until three weeks ago. The patient has been up and walking during the last six months.

Examination at the present time, November, 1929, revealed that there is no pain in the right leg. The left leg has never bothered him. There is an ulcer just above the medial malleolus about 1.5 by 2 cm. with some diffuse redness and edema about it. There is no edema or redness of the foot. There is some swelling of the foot at night but it disappears in the morning. The patient states that he has been much better since ligation of the femoral artery and vein and has had no pain since. He can put his weight on his foot and walk, which he could not do previously; he is well satisfied.

Case II.—L. G., a man aged fifty-six years, American born of pure English descent. His chief complaints were pain and ulceration of the left leg and foot; cold and cyanotic condition of the left foot; inability to walk.

Onset of illness started about fifteen years ago when, after walking a few blocks, a cramping, aching pain would start in the calf of the leg. It would pass away after resting. A similar pain started in the right leg eleven years ago with an ulcer over the tibia. There was no edema of the right leg. This ulcer never healed and the pain persisted until amputation of the right leg two years ago in another hospital. The left leg did not cause further trouble until about one year ago when the foot became cold and cyanotic and remained so. He stated that the right foot had never become cold. The left leg and thigh have felt tired and dead after a little exercise with shooting pains throughout. One month ago, pain and ulceration started together near the middle of the tibia on the outer side. Severe pains have prevented good rest at night.

The patient was a professional foot racer for seventeen years, from 1877 to 1894, and won 288 medals. He smoked from early youth. During his racing, he smoked four or five cigars a day. Since then he has smoked ten to fifteen cigarettes a day. He never drank much. He had gonorrhea in 1882. He has had no serious illnesses and was otherwise always well.

Family History: Father died of stomach trouble. Mother died of heart disease. Two brothers and two sisters are living and well.

The only previous operation was an amputation through the middle of the right leg two years ago.

Physical examination, August 10, 1927, revealed that his general condition was only fair; he was well developed but rather poorly nourished. The right leg was amputated about its middle. The left leg had a deep ulcer with necrotic base and edges over the anterior lateral side of the leg just above the middle. Tne leg was slightly red, cyanotic, and col 'below this point. There was no edema. He was unable to move the toes and foot without pain.

Laboratory Examinations: Wassermann negative; blood and urine normal.

First operation, August 31, 1927. Exposure of the femoral artery and vein in Hunter's canal. The femoral artery was completely occluded for several inches by an organized thrombus. A piece of the artery was resected through the thrombosed area and the vein was ligated with heavy silk.

Following this operation there was some immediate improvement. The leg became warm down to the foot and the patient could move the foot without pain. Four weeks after operation, the pain had recurred over the ulcer on the leg, and the lower leg was moderately cold. Seven weeks after the operation, October 23, 1927, when the patient had been out of bed about ten days, the leg had become badly congested to the knee with edema and severe pains in the foot and leg. The leg now appeared white on elevation and red on hanging down.

On November 2d, there was evidence of moist gangrene about the ulcerated area on the leg and the medial side of the foot was becoming necrotic from its middle to the big toe.

A second operation was done on November 2, 1927. Amputation of the leg about 6 inches below the knee was performed.

Fig. 114.—Specimen of thrombo-angiitis obliterans from Case II injected with bismuth oxychloride suspension. Spreading gangrene involved the big toe and area on the medial side of the foot and on the lateral side of the leg. There was an absence of filling of the posterior tibial and peroneal arteries in the leg with a rich collateral circulation about the ankle and a well-developed vascular bed.

Following amputation, gangrene of the stump below the knee developed for about 4 inches with sepsis. The lower half was edematous. The patient became very weak and toxic.

A third operation was performed on November 9, 1927. A guillotine amputation through the middle of the thigh was done.

Fig. 115.—Specimen amputated above the knee from a man, seventy-three years old, for spreading gangrene involving all of the toes. There was a recent thrombus occluding the popliteal artery. The main vessels were imperfectly injected due to extensive arteriosclerosis with calcification. The collateral circulation and vascular bed are diminished as compared to Fig. 114.

There was immediate improvement in his toxic condition and he seemed to be doing well. However, an extensive septic decubitus

and some local suppuration in the stump led to weakness and death on November 29, 1927.

Pathological Examination: The patent arteries were injected and roentgenograms made. This showed a rich vascular bed (Fig. 114). This may be compared to legs amputated for arteriosclerotic gangrene and thrombosis (Figs. 115, 116).

Fig. 116.—Amputation below the knee for dry gangrene of all the toes in a woman, sixty-three years old, in bed for one year previously due to a cardiac decompensation. There was a very scant vascular bed with small arteriosclerotic vessels difficult to inject.

The arteries in both the leg and thigh in Case II were dissected out.

1. The femoral artery was found to be completely thrombosed for some distance but the lumen was unobstructed for a short distance at the terminal end just above the enlarged collateral superior articular anastomosing branches of the knee.

2. The popliteal artery was almost completely thrombosed, but there was a fine lumen throughout.

3. The anterior tibial artery was incompletely obstructed for some distance by a thrombus, but there was a lumen.

4. The posterior popliteal was completely obliterated and contracted to pin size for most of its length. At the ankle it became patent again by a large collateral from the anterior tibial artery (Fig. 114). In the medial and distal half of the foot its branches became almost obliterated.

5. The perineal artery was contracted and the lumen obliterated.

Microscopical examinations were made of sections at various levels. This corroborated the gross findings and clinical diagnosis of thrombo-angiitis obliterans.

Case III.—This patient is a male, G. G., aged forty, American born of French descent. The first examination was made August 3, 1927.

His chief complaints at that time were pains, ulceration, and cyanosis of the right foot, developing rapidly in the previous month, with inability to use this foot.

The onset of the trouble began ten years ago when he noticed a limp in the left leg with numbness on walking. Gradually, this cleared up. Three years ago he had to stop work due to a recurrence. One year ago, pain and soreness developed between the toes of the left foot with ulceration. This became worse and the leg was amputated elsewhere about two months later. Pain and soreness developed in the right foot about one month ago with an ulcer between the first and second toes. This has become rapidly worse. He states that both legs always pained less when they were hanging down over the edge of the bed.

There has been no previous serious illness, and the only other illness was gonorrhea at twenty-eight years of age.

Family History.—His mother died at the age of sixty-nine years and the father died of "painters' colic" at the age of forty-five. Four brothers and five sisters are all dead of unknown causes. The patient is the youngest child.

The patient is a wood worker, standing all of the time since sixteen years of age. He has smoked a great many cigarettes daily since the age of fourteen years.

The only previous operation was amputation of the left leg and lower thigh about one year ago.

Physical Examination.—The patient was a rather short, heavy set man, fleshy and in good condition. The left leg was amputated in the middle of the thigh. The right foot was cold and cyanotic, as was also the lower half of the leg. There was no pulsation of the dorsalis pedis artery. There was an ulcer with a necrotic base between the first and second toes.

The first operation was done on August 10, 1927 and consisted of ligating both the femoral artery and vein with heavy silk. The artery was unusual for its small size, being only about one half or less its usual size. The walls were soft and pliable.

Following the operation there was warmth of the leg and foot noticed the next day. There was also some relief from the severe pain in the ulcer between the toes. There was no edema either before or immediately following operation. Improvement persisted for about one month, during which time the patient was up in a chair. At the end of this time the pain became worse and the gangrene began to spread from the two ulcerated toes to the foot and the patient became septic.

A second operation was done on September 18, 1927, at which time the right leg was amputated above the knee. The stump healed well. In April, 1928, the patient awoke with numbness and paralysis almost complete in the right hand and almost the entire arm. The neurological diagnosis was a cerebral thrombosis.

The arm is still useless but there is some motion and there is sensation present. He has some pains about the shoulder. This paralysis involves almost the entire brachial plexus. In January, 1929, he developed seizures resembling jacksonian epilepsy. These spells would start usually in the right side of his chest with contractions of the muscles and gradually spread to the right thigh and right side of the head and neck. Sometimes they would start with the head jerking to the right side.

These would last about five minutes and apparently were limited to the right side of the body. They would frequently fade away in his fingertips, but rarely in his neck. He would have these spells every week or two but never became unconscious. He has not been free from these seizures for over two months up the to present time, November, 1929.

DISCUSSION

Thrombo-angiitis obliterans has been designated as a disease usually occurring in young males characterized by an inflammation of the walls of the vessels and thrombus formation, with subsequent vascularization frequently terminating in gangrene. This condition may be differentiated from arteriosclerotic gangrene of the senile, in which the principle changes occur in the media and the adventitia. Lewis[8] found the average age of arteriosclerotic gangrene to be 66.2 years, while it was 54.4 years in the diabetic.

Thrombo-angiitis obliterans, however, occurs usually in relatively young males. It is rare in females. Meleney and Miller[9] found that about half of their group of 25 cases developed it in the fourth decade. Only 60 per cent of these cases used tobacco. However, it is usually recognized that this disease occurs more frequently in smokers. It seems to be more common in the Russian Jews and, according to Meleney and Miller, in the Chinese.

Silbert[10] stated that, usually, every case followed for a sufficient number of years has come to amputation. Seventy-seven per cent of one group of 155 patients observed came to amputation of at least one extremity within five years from the onset of symptoms. However, in more than four years of experience with treatment by means of repeated intravenous injections of hypertonic salt solution, based on 84 cases, at the time of the report only 12 per cent had come to amputation. Cessation of smoking, he believes, is the most important therapeutic measure.

An extensive collateral circulation has been shown by Meleney and Miller to develop below the site of a thrombosed main arterial segment. Improvement in the blood supply may

be due partly to the development of collateral circulation and partly to vascularization and canalization of the thrombosed main arterial segment.

The development of necrosis is thought to be due to the failure of collateral circulation to keep up with the extending obliterative thrombosis of the arterial lumen. While subsequent attacks may narrow the margin of safety from gangrene, naturally, the site of obliteration will modify the course of clinical symptoms.

The changes in thrombo-angiitis obliterans are patchy in character and are believed to result from inflammation. It may involve either large or small arteries or veins and is not necessarily an extending process from the periphery since the vessels are frequently found occluded above and free below. They may be occluded both above and below and free between. It appears evident that the disease frequently attacks the larger vessels before the vascular bed is disturbed.

Meleney and Miller observed frequent involvement of the nutrient arteries of the nerves and believed it might explain the severe pains associated with this disease. On the other hand, Bernheim and Sachs[11] found no thrombosis of vessels of the sciatic nerve no matter what disease process obstructed the main channel. They observed that the collateral circulation by way of the arterial branches along the sciatic nerves from the inferior gluteal artery may completely sustain the nourishment of the leg alone, in the presence of obstruction to its main vessels. Lipshutz[12] also found that the arteries accompanying the various nerves are among the most important vessels concerned in the formation of an adequate collateral circulation.

Barron and Linenthal[13] have emphasized the generalized character of the disease by pointing out the frequent involvement of the blood vessels of the brain, neck, thorax, and abdomen as well as of the extremities. They observed two typical cases of hemiplegia and one case of coronary thrombosis and reviewed 7 cases from the literature. They explain the vascular lesion in the brain as similar to that in the extremities and probably due to a partial occlusion with vascular spasm of the cerebral arteries

rather than to cerebral hemorrhage. These patients were under fifty years of age and their hemiplegia cleared up in so short a time that they believed it could not well have been due to a cerebral hemorrhage.

It is quite probable that a vascular lesion in the brain similar to that in the extremities was the cause of the partial hemiplegia with jacksonian epileptic seizures in Case III. However, the paralysis in this case was apparently permanent.

Lewis and Reichert have attempted to increase the collateral circulation in thrombo-angiitis obliterans by ligation of the femoral artery below the origin of the profunda femoris. They attempt to force the collateral circulation ahead of the advancing thrombosis. They reported ligation after this procedure in 7 cases. In four there was a distinct improvement. In two, ligation was performed after gangrene had developed and subsequent amputation was necessary. In one case death followed the operation with thrombosis involving the aorta, both iliacs, the subclavian, axillary, brachial, and internal carotid arteries extending within the skull.

Neill[14] reported one case having gangrene of the toes treated by ligation of the femoral artery below the profunda. There was local improvement until the patient suddenly died thirty-six days later, apparently with pulmonary embolism. A recent thrombus was found in the popliteal artery but apparently the main arteries of the leg were patent and the circulation of the foot was shown to be rich from roentgenograms made after the injection of the specimen.

Jennings[15] presented a case with local necrosis at the base of the previously amputated great toe in which he ligated the femoral artery below the profunda. There was immediate gangrene extending to the knee requiring a thigh amputation.

Ligation of the accompanying vein together with the artery has been shown experimentally and clinically to result in a better blood supply, a diminution of gangrene and a greater increase in functional ability than when the artery alone was ligated. It has further been shown experimentally that this results in an increase in residual arterial pressure, in the venous pressure, in

the peripheral arterial circulatory bed and perhaps also in an increase in the capillary circulation. There is, probably, a fall in blood flow associated with the rise in peripheral blood pressure.

Hamilton Drummond[16] found experimentally that by carefully selecting two loops of ileum a definite ring of gangrene developed in a majority of cases in which the artery alone was ligated while the segment in which both artery and vein were ligated showed little or no change.

Van Kend[17] observed by plethysmograph tracings on animals that a slight rise in the blood pressure in a limb follows the application of a ligature to a main vein after previous ligature of the artery. He concluded that physiologically leaving the main vein viable after occlusion of the main artery of a limb diminished what may be called the residuary blood pressure maintained by the collateral circulation. If the contribution of the collateral circulation is allowed to remain with the main vein intact it is natural that the residuary blood pressure should fall.

Ligation of the main vein in addition to the artery would maintain the blood in longer contact with the tissues. This would prevent a too ready exit of the blood from the already diminished arterial flow and also prevent an undesirable reservoir for stagnation.

Brooks and Martin[18] observed that the temperature of an extremity throughout its whole distal portion fell more after ligation of a vein and artery than after ligation of an artery alone. They interpreted this as a fall in the volume of the blood flow. Furthermore, they found that the intravascular pressure in both arteries and veins was decreased by ligation of an artery alone and that obstruction of the vein caused a rise. The blood pressure in the veins rose more, proportionately, than it did in the arteries. They believed that the more complete the venous obstruction, the more nearly intravascular pressure was raised to that of the artery and the lessened blood flow.

They observed a marked lessened occurrence of gangrene in extremities where both artery and vein were ligated, $33\frac{1}{3}$ per cent, than where the artery alone was ligated, 71.5 per cent. Their conclusions were that the gangrene was not the result of

the amount of blood flowing through the vessels distal to the obstruction necessarily, and that there might be a minimum of intravascular pressure for exchange of nutrient substances from within the vessels to the tissues.

Holman and Edwards[19] in sacrifice experiments observed that the volume flow after arterial ligation was increased by ligation of the vein, the increase being dependent upon the site of the obstruction. Ligation of the vein proximal to the arterial branches furnishing collateral circulation produced a sharply increased blood pressure over ligation of the vein at the same level. They observed that gangrene of the extremities occurred in only 7.1 per cent of animals in which the vena cava was ligated simultaneously with the common iliac artery as compared to $33\frac{1}{3}$ per cent in which ligation of the vein was done at the same level.

By comparing animals studied over a long period of time after experimental ligations, Pearse[20] observed that the increased residuary arterial blood pressure and blood flow which occurred following ligation of the artery alone was a transient phenomenon, diminishing gradually until at the end of three weeks they were the same. However, Pearse found that ligation of the artery and vein was always followed by a richer collateral vascular bed than that following obstruction of the artery alone and believed that this was the fundamental cause of the diminished gangrene and improved function. Ligation proximal to the site of ligation of the artery according to Holman's[21] principle resulted in a better vascular bed than any other procedure. He concluded it was not the increased arterial pressure that improved the results of concurrent artery and vein ligation over that of the artery alone, since it was only such a transient phenomenon, but that it might be a factor in the increased arterial vascular bed.

On the other hand, Theis[22] believed that the collateral circulatory bed was better developed in those instances in which the artery and vein were occluded, only if the determination was made immediately afterward. After a period of three weeks he concluded that there was a richer vascular bed in those experimental animals in which the artery alone was occluded. Theis

observed an increased volume of blood flow in the extremities of animals after several weeks following ligation of the artery alone, as compared to both the artery and vein.

Brooks, however, believed that the size of the blood vessels was not a good index of the actual volume flow through the tissues and that the condition of the arterial bed three weeks after the arterial occlusion was not necessarily important in gangrene. Brooks[23] cautioned against the routine simultaneous ligation of the vein with the artery, explaining that the immediate beneficial effects must be balanced with the possible remote ill effects of chronic venous stasis. Certainly, experimental observations three weeks after any operative procedure on the artery should not be considered final, since it is a well-known fact that, clinically, a periarterial sympathetic nerve interruption will persist that long.

Clinically, Oppell[24] observed good results by occlusion of the popliteal vein in the treatment of 6 cases of senile gangrene of the foot. McNealy,[25] after failure to produce any appreciable improvement in cases of thrombo-angiitis obliterans by a periarterial femoral sympathectomy alone, combined sympathectomy with ligation of the main vein. No improvement was noted but he apparently did not ligate both artery and vein for this condition. Morton and Pearse,[26] after ligation of the popliteal vein in cases of thrombo-angiitis obliterans and arteriosclerotic gangrene, found improvement in the majority with an elevation of temperature of the extremity. While this seemed at variance with the finding of Brooks, who observed a fall in the temperature of a limb upon occluding a vein having its main vein obstructed, they explained that in their cases, usually, the artery was only partially occluded by disease. Van Gordon[27] observed improvement after high ligation of the vein in a number of cases of thrombo-angiitis obliterans.

Makins in 1913 observed that proximal ligation of the femoral artery in cases of arteriovenous aneurysm was followed in a large proportion of instances by gangrene of the limb, while excision of the implicated segments of both artery and vein gave consistently good results.

During the World War he observed that gangrene resulted in only 14 per cent of 71 cases in which both the artery and vein were ligated, as compared to 29 per cent of 101 cases in which the artery alone was ligated. Makins' practice of simultaneous ligation of both artery and vein was adopted by others.

Tuffier[28] introduced a paraffin coated silver tube into the ends of severely injured main arteries. This permits collateral circulation to establish itself during the gradual occluding thrombosis in the tube, which takes from a few hours to rarely as long as ten days.

It must be remembered that the blood supply to a part is always dependent upon a primary physiologic demand of the tissues. However, abnormal nerve reflexes, arteriosclerosis, and diseased conditions may alter the normal ability of healthy blood vessels to supply the necessary amount of blood.

While considerable emphasis has been placed on the finding of a rich vascular bed in cases of thrombo-angiitis obliterans, this might be expected since they are comparatively all men, many of the athletic type, under forty-five years of age. The pathologic findings of the blood vessels following a thrombosis in the senile may also show areas of round-cell infiltration in the wall, and later canalization. Although calcification is usually extensive in the senile cases developing gangrene it may also be demonstrated in some typical cases of thrombo-angiitis obliterans.

It is difficult to compare results of sudden vascular occlusion obtained either in sacrifice animals of those observed over a long period of time, with a chronic obliterative process in man. Similarly, results obtained in the human following ligations or sudden occlusion of healthy blood vessels should not be compared to the gradual occlusions in thrombo-angiitis obliterans frequently extending over years with their patchy inconstant location to the main trunk or periphery of either the arteries or veins.

In Case I, observed twenty-seven months after ligation of the femoral artery and vein in Hunter's canal, there has been a permanent relief of pain, followed by healing of two gangrenous

areas on the foot, although ulceration temporarily recurs on the ankle. In the other 2 cases, which were more advanced and upon whom previous amputation of the opposite leg had been done, temporary improvement was followed by extension of gangrene and amputation.

Any procedure which may relieve pain and avoid amputation, even though normal function is not obtained, should be considered in the treatment of this condition. Therapy must be directed to general treatment in checking the disease, which apparently is not a local, but a general process, in its early stages, and not merely to the relief of pain and retarding gangrene. The elimination of focal infection, the establishment of a balanced diet, and good hygiene are encouraging fields in this direction.

Results from concomitant ligation of the artery and vein may be valuable under certain combinations of obliterating lesions, and even harmful in others. Whether ligation of the vein together with the artery will materially aid other methods of treatment in thrombo-angiitis obliterans, will probably depend upon further experimental reasearch and clinical usage in carefully selected cases.

BIBLIOGRAPHY

1. Phillips, H. B.: Roentgen Therapy of Neurocirculatory Diseases, Med. Jour. and Rec., New York, 128, 627, December 19, 1928.
2. Allen, E. V.: The Results of Lumbar Ganglionectomy in Thrombo-angiitis Obliterans, Proc. of Meetings of Mayo Clinic, 3, 303, October 17, 1928.
3. Brown, G. E.: Treatment of Peripheral Vascular Disturbances of the Extremities, Jour. Amer. Med. Assoc., 87, 379, August 7, 1926.
4. Allen, A. W., and Smithwick, R. H.: Use of Foreign Protein in the Treatment of Peripheral Vascular Diseases, Jour. Amer. Med. Assoc., 91, 1161, October 20, 1928.
5. Allen, E. V., and Meyerding, H. W.: Surgical Procedure in Obliterative Vascular Disease (Thrombo-angiitis Obliterans), Surg., Gynec., and Obst., 46: 260, Feb., 1928.
6. Lewis, D., and Reichert: The Collateral Circulation in Thrombo-angiitis Obliterans, Jour. Amer. Med. Assoc., 87, 302, July 31, 1926.
7. Makins, G. H.: On Gunshot Injuries to the Blood Vessels, published by William Wood and Company, New York, 1919.
8. Lewis, Dean: Spontaneous Gangrene of the Extremities, Arch. of Surg., 15, 613, October, 1927.

9. Meleney and Miller: A Contribution to the Study of Thrombo-angiitis Obliterans, Ann. of Surg., 81, 976, May, 1925.
10. Silbert, S.: Studies on Thrombo-angiitis Obliterans (Buerger), Jour. Amer. Med. Assoc., 89, 964, September 17, 1927.
11. Bernheim, B. B., and Sachs, L.: Notes on the Collateral Circulation in Blood Vessel Diseases of the Lower Extremities, Ann. of Surg., 86, 417, September, 1927.
12. Lipshutz, B.: Injuries of the Large Arteries, Surg., Gynec., and Obst., 46, 63, January, 1928.
13. Barron, M. E., and Linenthal, H.: Thrombo-angiitis Obliterans; General Distribution of the Disease, Arch. of Surg., 19, 735, October, 1929.
14. Neill, T. E.: Ligation of the Femoral Artery Below the Origin of the Profunda Femoris in the Treatment of Obliterative Endarteritis of the Leg, Ann. of Surg., 86, 425, September, 1927.
15. Jennings, J. E.: Sympathectomy for Thrombo-angiitis Obliterans, Ann. of Surg., 87, 954, June, 1928.
16. Drummond, Hamilton: Quoted by Makins.
17. Van Kend, M.: Quoted by Makins.
18. Brooks, B., and Martin: Simultaneous Ligation of Vein and Artery, Jour. Amer. Med. Assoc., 80, 1678, June 9, 1923.
19. Holman, E., and Edwards, M. E.: A New Principle in the Surgery of the Large Arteries, Jour. Amer. Med. Assoc., 88, 909, 1927.
20. Pearse, H. E.: An Experimental Study of Arterial Collateral Circulation, Ann. of Surg., 88, 227, August, 1928.
21. Holman, E.: Observations of the Surgery of the Large Arteries, Ann. of Surg., 85, 173, 1927.
22. Theis, F. V.: Ligation of Artery and Concomitant Vein in Operations on the Large Blood Vessels, Arch. of Surg., 17, 244, August, 1928.
23. Brooks, B.: Surgical Applications of Therapeutic Venous Obstruction, Arch. of Surg., 19, 1, July, 1928.
24. Oppell, W. A.: Zentrabl. f. Chirurg., 31, 1241, 1913.
25. McNealy, R. W.: Injuries to Blood Vessels and Their Management, Illinois State Med. Jour., 48, 48, July, 1925.
26. Morton, J. J., and Pearse, H. E., Jr.: Temperature Effect of Popliteal Vein Ligation in Thrombo-angiitis Obliterans and Arteriosclerosis, Ann. of Surg., 88: 233, Aug., 1928.
27. Van Gordon, G. W.: High Vein Ligation in Thrombo-angiitis Obliterans, Ann. of Surg., 90, 88, July, 1929.
28. Tuffier: Quoted by Makins.

CLINIC OF DR. GATEWOOD

PRESBYTERIAN HOSPITAL

APPENDICITIS IN OLD AGE

DESPITE the enormous literature on appendicitis the recent statistical reports show an increasing mortality. The reasons for this are not at once apparent, but in looking over our cases, we have been struck by the fact that the mortality occurs chiefly at the two extremes of life. The seriousness of appendicitis in children has been well recognized, but there has not been so much discussion about appendicitis in the aged. Maes,[1] discussing the subject at the Louisiana Medical Society, gave a mortality of 29 per cent in patients over fifty years of age. E. M. Fitch[3] in 1928 reported the results of a questionnaire covering 6548 cases. The average mortality rates of the institutions replying to his queries varied from 1.9 to 4 per cent. In the patients over fifty years of age the death rate varied from 19 to 50 per cent. In his series at the Claremont (N. H.) Hospital, the mortality was 21.6 per cent in patients between fifty and sixty, and 54 per cent in patients between sixty and seventy. Part of this high mortality in the aged is attributable to the lowered resistance of the patients. Much more, however, can be explained upon the basis of errors in diagnosis and upon the pathology peculiar to these older individuals. Lehmann[3] reported 6 cases from the Versorgungsheim in Lainz, calling attention to this atypical picture; and Erdheim, in his pathologic conferences at the same institution, has frequently demonstrated autopsy specimens of old people dying of unrecognized appendicitis with resultant peritonitis.

The diagnosis, even when a typical history is obtainable, is frequently missed because of the rarity of the disease. Maylard[4]

found, in 1000 consecutive cases of appendicitis observed in the Victoria Infirmary, Glasgow, that only 1.5 per cent occurred in patients between sixty and seventy years of age and only .3 per cent occurred in patients over seventy. Self-treatment before a physician is consulted often adds to the confusion and, in the majority of cases, a typical history is not obtainable. Rarely is a history of preceding attacks obtained. Older people complain less of symptoms than younger individuals, probably because of a lowered sensitiveness. Vomiting is less often an early symptom and nausea may be in the background. The elevation of temperature which is a sign of systemic reaction to infection is frequently absent and the pulse rate is rarely over 100. The temperature in none of Lehmann's 6 cases was above 100 F. The leukocyte count is usually elevated from 12,000 to 15,000 and this may be a clue to the diagnosis. Many patients are not seen until peritonitis is well developed and pain and tenderness are generalized. The abdominal distention of paralytic ileus suggests intestinal obstruction and valuable time may be lost in attempting to locate a suspected neoplasm.

The pathology is characteristically acute. Usually there are very few adhesions; peritonitis is widespread with little tendency to walling off. The appendix is frequently gangrenous throughout, the gangrene extending to the mesoappendix and to the walls of the cecum, making it very friable, and appendectomy correspondingly difficult. This is the type of appendicitis ferquently followed by pylephlebitis and liver abscess.

It is probable that many of these infections are embolic, and arteriosclerosis may play a part in the retrograde thrombosis so frequently observed. This type of infection is likely to be followed by thrombosis of the vessels of the abdominal wall paving the way for embolism or infarct, and the resultant postoperative pneumonia increasing the mortality.

The patient whom I wish to demonstrate is a man seventy-seven years old who has been sick for two days. His chief complaint is abdominal pain, which has been steadily increasing in severity. The pain was generalized at the onset. Yesterday it seemed to be chiefly over the right side, but today it is again

generalized. The patient has been belching and has had some nausea, but no vomiting. The pain at the present time seems to be intermittent, lasting from one to two minutes with partial relief for four or five minutes. His general health is otherwise good except for some hesitancy in starting his urine, and occasionally some dribbling. He has had nocturia for two or three years. His past history is negative except for a somewhat similar attack about six weeks previously which came on rather suddenly and abated gradually.

On examination the patient appears acutely ill. He is a well-preserved old gentleman. The abdomen is distended and tympanitic. There is no dulness in the region of the bladder. There is marked tenderness over the entire abdomen, seemingly most marked over McBurney's point. With a stethoscope placed on the abdomen, a distinct gurgling sound can be heard at frequent short intervals. The patient's temperature is 98 F., his pulse 88, and respiration 20. His leukocyte count is 14,000. Urine is negative except for a few leukocytes. Owing to the uncertainty of the diagnosis I have had the patient taken to the fluoroscopical room and a barium enema given, the results of which are of considerable importance in our preoperative diagnosis. Before the injection was made numerous distended loops of bowel could be made out under the fluoroscope. When the barium was given, the colon filled rapidly without evidence of obstruction. The patient complained of pain as soon as the enema reached the cecal region and the cecum did not completely fill (Fig. 117). The appendix was visualized and pressure over it produced pain. The patient then expelled the enema so that there has been but slight delay in preparing him for operation. From the history and physical findings, I believe we can make the preoperative diagnosis of acute appendicitis with ileus.

Operation.—Under novocaine infiltration anesthesia the abdomen has been opened. Much free turbid fluid is present. The intestines are distended and red, showing diffuse peritonitis. The appendix is gangrenous and also the cecum immediately surrounding it. In removing the appendix, we are having some difficulty on account of the friability of the indurated cecum.

As much fluid as could be readily removed by gentle mopping and by the use of the suction apparatus has been withdrawn. The wound is now partially closed and drainage instituted. It has been necessary to use gas for delivery of the appendix and for part of the closure. We could find no evidence of obstruction. Occasionally gurgling is heard in paralytic ileus, especially when a cathartic has been given.

The after-care here will consist first in putting the patient in a semisitting posture with the head of the bed elevated. In

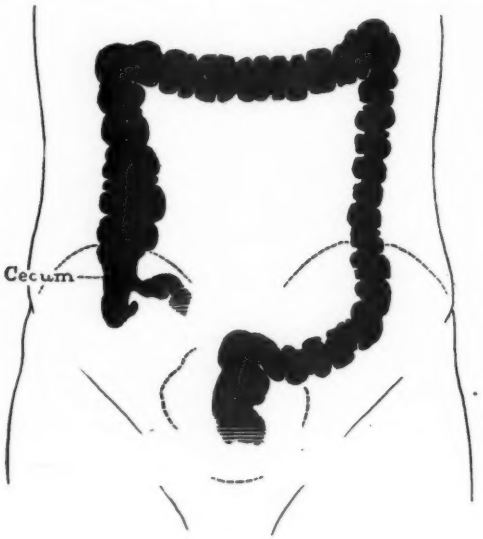

Fig. 117.

this way, he can be kept reasonably comfortable without being doubled up. This will allow gravity to keep the infected fluid localized to the lower abdomen, as well as permitting better lung expansion, by keeping the weight of the intestines away from the diaphragm. If he does well, the patient will be gotten up in a chair about the fifth day. Second, he will be given normal salt subcutaneously by the Woodyatt continuous method in sufficient quantity to prevent dehydration. Stimulating

enemas must be used with caution, owing to the difficulty in closing over the appendix stump, but small magnesium sulphate-glycerin injections will do no damage. Morphine should be used only in amounts necessary to make the patient comfortable.

Comment.—This patient should have been seen and operated upon at least twenty-four hours sooner. His history would have been considered typical in a younger man and his physical findings yesterday probably were definite enough had he been carefully examined. Old people stand surgery reasonably well in the absence of severe infection, and it behooves us to keep the possibility of appendicitis in mind whenever dealing with an acute abdomen regardless of the patient's age. Medical management is more apt to doom these old people than younger individuals, owing to the failure of the infection to localize. If we are to reduce our appendicitis mortality, we must make our diagnosis in this group early, and institute operative therapy.

BIBLIOGRAPHY

1. Maes, U.: Appendicitis in the Aged, New Orleans Med. and Surg. Jour., 78, 117, September, 1925.
2. Fitch, E. M.: Appendicitis in People Over Fifty Years of Age, New England Jour. Med., 198, 348, April 5, 1928.
3. Lehmann, H.: Appendicitis in Old Age, Wien. klin. Wchnsch., 40, 995, August 4, 1927.
4. Maylard, A. E.: Sex and Age Incidence in Appendicitis, Brit. Jour. Surg., 8, 189, October, 1920.

INFECTED CYST OF THE URACHUS

THIS patient, a married woman, twenty-six years old, comes to me on account of discharge from the umbilicus and obesity. This discharge has been present for several weeks. It has been a reddish, semipurulent, offensive-smelling fluid. Her history is otherwise negative.

On examination the patient weighs 240 pounds. Her abdomen is very rotund, making examination difficult, but except for an infantile uterus, no pathology can be felt within the abdominal cavity. In the base of a rather shallow umbilicus is a small reddish elevation from which pus exudes when pressure is made over the lower abdomen. Introducing a probe, I find that there is a sinus at least 5 cm. deep. On careful questioning, there has been nothing like fecal matter discharged from this sinus. Defects in the closure of the embryonic abdomen are always of interest. They may vary all the way from complete eventration to small sinuses such as we find here. Evidence of some of the minor anomalies may only be found at autopsy. This is the typical history for an infected cyst of the urachus, although a remnant of the omphalomesenteric duct must be considered. Persisting as a part of Meckel's diverticulum, omphalomesenteric duct remnants usually discharge fecal material. Cysts may occur at any point in the remnants of the allantois, or the entire urachus may remain patent and discharge urine from the apex of the bladder. While this condition usually is found in the newborn, Weiser[1] reported a case in which a man, aged seventy-three, developed a urinary fistula at the umbilicus, and Cullen, in his book on the Umbilicus and Its Diseases, cites a number of cases from his large personal experience and the literature in which fistulae have developed during adult life. Like pilonidal cysts, if not discharging at birth, these cysts

[1] Weiser, W. R.: Cysts of the Urachus, Ann. Surg., 44, 529, 1906.

frequently cause no symptoms until adult life, and then usually as the result of an infection. Cullen shows by graphic diagrams the various possibilities which may be found, and which should be kept in mind whenever a tumor mass is felt in the abdomen either at or below the umbilicus.

Owing to the patient's size I am operating upon her under local anesthesia. Having infiltrated the entire surrounding area

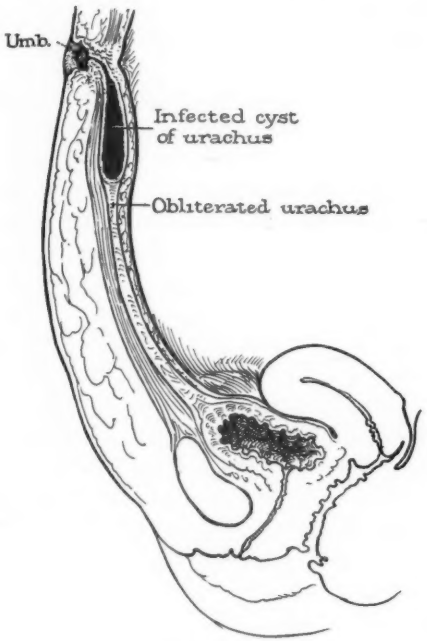

Fig. 118.

with ½ per cent procaine, I am excising the entire umbilical depression. With a uterine probe introduced into the sinus I am able to dissect out the entire tract without opening it. It leads through the linea alba and downward about 5 cm. It is extraperitoneal and follows the anterior abdominal wall in the midline (Fig. 118). Closure is made by overlapping the linea alba

from above downward by three mattress sutures, making a typical Mayo umbilical hernia repair.

Postoperative Note.—Sections of this tract show a transitional type of epithelium, some round-cell inflammation, and nonstriated circular muscle fibers, confirming the operative diagnosis of infected cyst of the urachus.

CLINIC OF DR. GEORGE M. CURTIS

ALBERT MERRITT BILLINGS HOSPITAL

INTRATHORACIC GOITER

INTRATHORACIC goiter is particularly a problem in regions where goiter is severely endemic, and where its incidence, as a consequence, is greater. It must be kept in mind, however, even where less frequent. This applies especially to those forms with moderate symptomatology, due either to pressure or to toxic effects. Roentgenological study affords the surest means of diagnosis. Intrathoracic goiter is, as a rule, nodular. It is rare to discover one of the diffuse type. The tendency of nodular goiters to grow, to become cystic, to become acutely enlarged by hemorrhage, and to develop carcinoma is considered an indication warranting their removal. These changes are accentuated in importance when the goiter lies among the vital structures of the superior mediastinum.

The classification of intrathoracic goiters is at present confused. In general, three forms are recognized: First, the *struma profunda*, or deep goiter, associated with a low-lying thyroid. In this condition, the *thyroptosis* of Kocher, the lower poles or isthmus descend into the superior aperture of the thorax. Upon swallowing the gland is elevated and the lower poles may be palpated over the clavicles or sternum. The second type is the *partial intrathoracic goiter*. Associated with a cervical enlargement, a goitrous process extends into the thorax. There thus exists both a cervical and an intrathoracic portion of the goiter. It is in this type that the greatest confusion exists, although a part of the goiter remains intrathoracic even on swallowing. The third type is the rarest and, at the same time, the most definite. In this the entire goiter lies within the thorax.

When associated with practically no goiter in the neck the designation becomes clear, as in the case presented this morning as an example of *total intrathoracic goiter*. To some clinicians this is the only true intrathoracic form.

Intrathoracic goiter develops as a rule from the lower thyroid gland. It is rare as an accessory thyroid. As the nodular mass grows it pushes ahead of it cervical, and later thoracic fascia, and may thus form a condensed envelope as it migrates into the superior mediastinum. As a rule it maintains its blood supply from above and does not ordinarily become markedly adherent to its envelope. A consideration of this fascial envelope is of the greatest importance in the surgical removal. Intrathoracic goiter is essentially the "ingrowing goiter" feared by the laity.

Intrathoracic goiter is not always accompanied by even moderate symptoms of compression or thyrotoxicosis. This applies particularly to those cases without a cervical goiter, in which the growing totally intrathoracic lobe expands insidiously among the pliant structures of the superior mediastinum. In fact, it may be masked in situation as well as symptomatically. The case which I am presenting this morning illustrates this type. In this instance there were almost no symptoms during the development of the disease, and no visible or palpable goiter.

Case I. Masked Total Intrathoracic Goiter.—One of our medical students brought her mother aged fifty-nine to the clinics for a general examination, particularly owing to a gastro-intestinal disturbance. She was aware of a rather indefinite epigastric mass, and of a vague sense of fulness after meals; there had also been a recent loss of appetite, but no weight loss. She feared cancer even in the face of the meager signs and symptoms. Some dyspnea upon exertion and chronic choking attacks were also noted. The latter, however, had appeared to follow the passage of food or saliva into the larynx, and had been attributed to some defect in the epiglottis. The patient was not aware that she had a goiter, nor was there goiter in her family history.

Examination revealed a healthy, active woman with good

color and an abdomen essentially negative to palpation. Fluoroscopy showed a frank, persistent defect in the duodenal bulb, with which was associated a rather indefinite mass. The films, however, were not conclusive of duodenal ulcer. During the fluoroscopy the roentgenologist, Dr. C. S. Capp, discovered a striking soft tissue shadow in the upper thorax. This was thought at first to be an esophageal pouch. Swallowing barium, however,

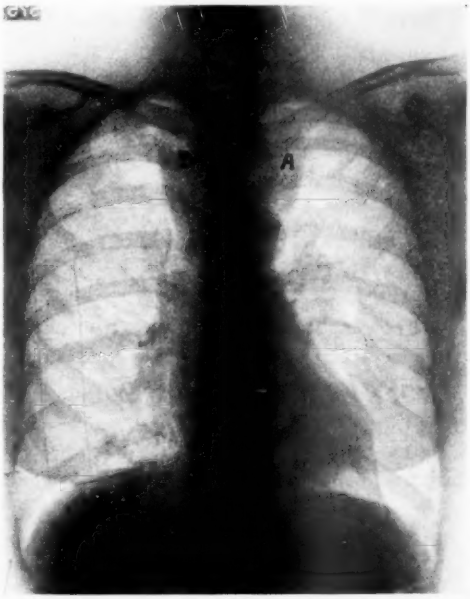

Fig. 119.—Case I. Preoperative roentgenogram, total intrathoracic goiter: A, Goiter shadow; B, compressed and deviated trachea.

soon revealed that the esophagus was narrowed and pushed far to the right at the first costal cartilage. Films disclosed a large, sharply outlined, soft tissue shadow (Fig. 119, A) lying well forward in the superior mediastinum, and extending into the second interspace, beyond the aortic arch. The trachea was compressed to about one third its normal diameter and deviated to the right throughout its entire extent (Fig. 122). The great-

est displacement was at the level of the first costal cartilage (Fig. 119, B). The lungs were clear and the heart shadow appeared normal (Fig. 119).

The nature of the intrathoracic mass was next considered. The Wassermann and Kahn were negative. The patient was fifty-nine years old. There was no lymphadenopathy. The blood picture was normal, with a 33 per cent lymphocytosis. The shadow was distinctly anterior to that of the aortic arch and made a definite angle with it (Fig. 119). There was no palpable cervical goiter; however, there was definite fulness under the inner end of the left clavicle, and in the suprasternal notch. The larynx was turned slightly to the right and moved but little on swallowing. The trachea was definitely deviated. There was percussion dulness over the superior mediastinum, particularly to the left. The basal metabolic rate was — 11, with the pulse at 60, respirations at 12 and temperature at 98.2 F. in the basal state. The blood pressure was 136/92 and the radial pulses were equal. There was no exophthalmos, tremor, or tachycardia, or no engorgement of the cervical or brachial veins. The vocal cords were normal. The urine was negative. By orthodiagram the heart was 14 per cent oversize. The electrocardiogram revealed a normal mechanism.

A second fluoroscopy with Dr. P. C. Hodges demonstrated that the intrathoracic shadow was continuous into the lower left neck, and confirmed the observation that the mass remained fixed on deglutition. Swallowed barium bougies were again deviated to the right and passed posterior to the main mass. It was then elicited that the patient had had choking attacks intermittently for many years and that these were occasionally spontaneous. There had also been some dysphagia and occasionally a sense of suffocation. A diagnosis was consequently made of total intrathoracic goiter with marked deviation and compression of the trachea and esophagus, without thyrotoxicosis.

It is important to reemphasize at this point that intrathoracic goiter is as a rule nodular. Consequently it is more frequently seen in clinics where nodular goiter prevails. Nearly a quarter of all the goiters removed in de Quervain's Clinic in Berne be-

tween October 1, 1925 and December 31, 1926, were classed as intrathoracic. But two of these 66 intrathoracic goiters were diffuse in character. Higgins[1] reports that 97 per cent of 100 intrathoracic goiters removed in the Cleveland Clinic were nodular. Nodular goiters are particularly predisposed to a number of degenerative changes, notably in the walls of the arteries. Jores[2] has described and figured the early weakening of the arterial walls due to hyaline changes, calcification and fragmentation of the internal elastic membrane. These may occur in nodular goiters without a generalized arteriosclerosis. Atheromatous plaques have been observed in the thyroid arteries of the newborn,[3] and arteriosclerosis in the thyroid arteries of infants.[4] It is a common observation that the majority of nodular goiters show hemorrhage either grossly or microscopically. Hemorrhage is dangerous in that it may lead to further tracheal compression and suffocation. Nodular goiters commonly undergo cystic degeneration. Enlargement of these cysts may increase the compression symptomatology. Malignancy develops most frequently in association with nodular goiter. Nine per cent of 1544 goiters operated in Berne between 1911 and 1922 were malignant (Wegelin[6]). By coincidence the one case of carcinoma of the thyroid which we have observed among 125 thyroidectomies at the Billings Hospital was associated with a large cystic intrathoracic goiter, No. 8513.

The mortality associated with the removal of intrathoracic goiter is low. Matti[5] has recently reported 219 successive cases without a mishap. In view of our increasing knowledge of this disease it seemed wiser to advise removal of the intrathoracic goiter, even if the symptoms were minimal, rather than leave the patient to the nodular possibilities. In fact, in advising surgery we considered prophylaxis as an important feature.

After a preoperative $\frac{1}{4}$ grain of morphine the entire operation was done under local anesthesia; $\frac{1}{2}$ per cent novocaine with 8 drops of adrenalin per 100 cc. To facilitate exposure, the sternohyoid and sternothyroid muscles were cut high on the left side, and reflected. It was not necessary to cut the sternomastoid. The thyroid gland was but little enlarged and non-

nodular. Extending from the inferior pole of the left lobe, and connected to it by a short narrow stalk, was an ovoid, nodular mass which, when explored, proved to be wholly intrathoracic. In fact, the situation discovered recalled that figured by Wegelin.[6] The attached lower left pole was first freed from the left lobe and brought forward. Complete hemostasis was secured by

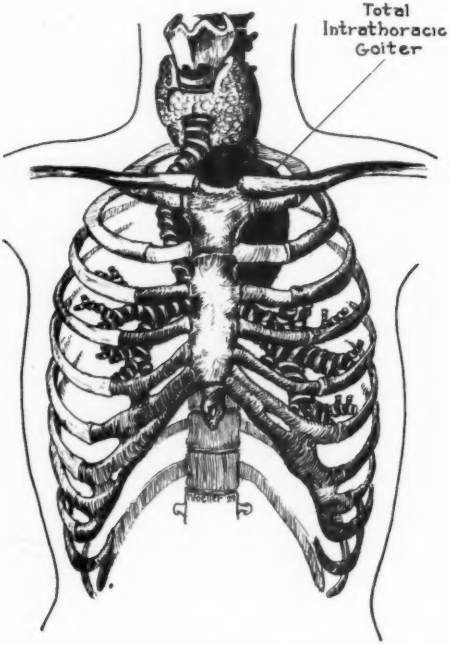

Fig. 120.—Case I. Semidiagrammatic drawing illustrating the position of the goiter and deviated trachea.

fixation ligatures. Neither the superior nor the inferior thyroid arteries were ligated. The proper plane of cleavage was determined and the ovoid lobe was separated from its loose attachments by blunt fingertip dissection. It was not particularly adherent to the surrounding condensed fascia. A large needle was plunged into the upper portion of the mass and about 5 cc.

of blood-tinged fluid was aspirated. Cutting off the main blood supply from above, as well as aspiration of any cystic fluid facilitates the removal of an intrathoracic goiter by decreasing its size. Clamps were then placed around the upper cut margin and by means of fingertip manipulation, traction and rotation, and the conscious patient's cooperation in increasing the intra-

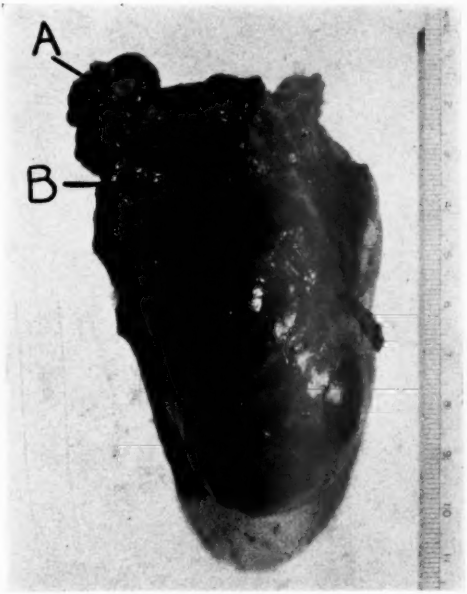

Fig. 121.—Case I. Excised total intrathoracic goiter, from the right. A, Detached lower left pole of the thyroid; B, collapsed cystic nodule which was aspirated.

thoracic pressure, the goiter was obstetrically delivered over the inner end of the clavicle. But one hemostat was necessary to clamp a single minor vascular attachment. The subclavian artery lay posterior and to the left, likewise the common carotid. The tip of the goiter extended below the origin of these arteries from the aortic arch, and lay 8 cm. below the upper margin of the clavicle. The left innominate vein, however, lay ahead of

the nodule. The unbroken membrane lining the cavity which remained was composed of the condensed fasciae surrounding the goiter. It was fairly smooth and tough in consistency. The trachea was sharply deviated to the right and anteriorly, and was compressed. There was no attack of choking or of respiratory embarrassment during the luxation of the goiter. The voice

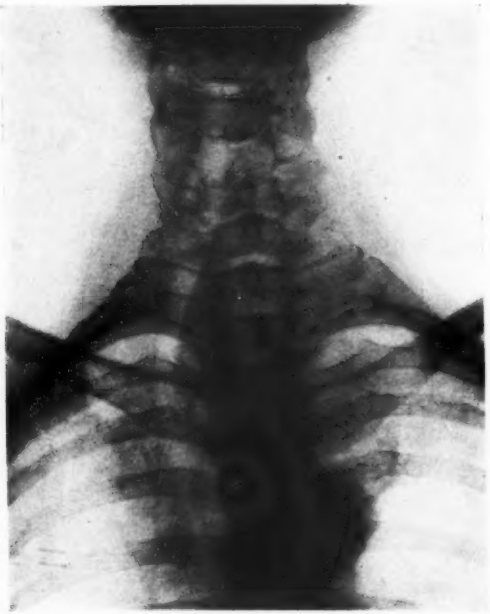

Fig. 122.—Case I. The preoperative compression and deviation of the trachea.

was readily controlled. There was no particular pain during the luxation. Novocaine was injected only into the skin and muscles. A soft cigarette drain was left in the deep cavity and extended out of the lateral angle of the wound. A Penrose drain was inserted to the region of the lower left pole and passed out of the middle of the incision.

Convalescence was uneventful. The drains were removed on

the second and third days. There was but a moderate amount of serous drainage which was sterile bacteriologically. The patient left the hospital on the sixth day. The wound healed without further drainage. The basal metabolic rate three weeks following operation was plus one with the pulse at 64, the temperature 98.2 F., and respiration at 9 in the basal state. The blood pressure was 120/60.

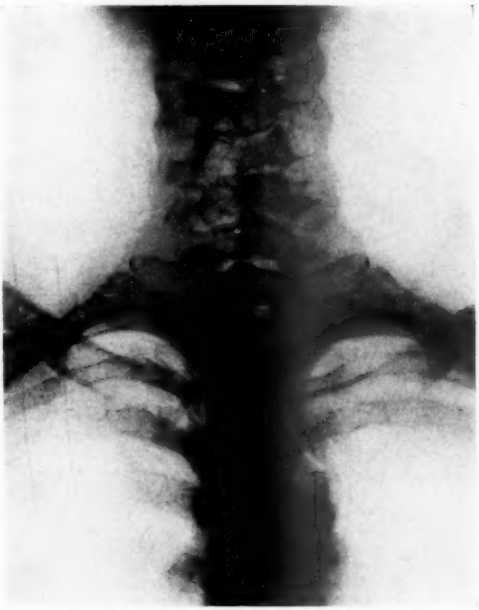

Fig. 123.—Case I. The trachea twenty-one days after removal of the intrathoracic goiter.

The excised lobe (Fig. 121) was ovoid, with some anteroposterior flattening and a groove on the right anterolateral surface due to the trachea (Fig. 120). It weighed 82 Gm. and measured 9 by 6 by 4 cm. It was attached at one margin of its broadened base, which contained a partially cystic nodule, to the lower pole of the left lobe (Fig. 121, A). The broad nodular

base and the narrowed attachment doubtless account for its fixation in the superior thoracic aperture on swallowing. One cystic nodule appears in frontal section. This measures 3 by 2.5 by 2 cm. and is visible at the center of Fig. 121. This contains extensive hyaline and a considerable area of recent hemorrhage. Another nodule, slightly smaller and solid, presents numerous colloid masses. Sections reveal a nodular colloid

Fig. 124.—Case I. The return of the trachea to normal position three months after operation.

goiter. There are several areas of recent hemorrhage, as well as extensive evidence of old. Numerous small flecks of calcification are present. The pathologic diagnosis is nodular colloid goiter with cystic degeneration, recent and old hemorrhage, and calcification.

It has been of particular interest to us to follow roentgenologically, in a number of cases, the changes occurring in the de-

viated and compressed trachea, subsequent to the removal of the offending goiter. The roentgenograms demonstrating the restitution to normal shape and position in this instance are presented in Figs. 122–124. They need but little added description. In three weeks a striking increase in diameter had already occurred and the trachea was nearly median. Oblique films added no further information. In eight weeks the trachea was indistinguishable from normal. During the past year we have observed the same restitution following the removal of a large nodular and cystic partial intrathoracic goiter, No. 7788. There is an associated disappearance of the related symptoms.

The majority of intrathoracic goiters occur on the left side, as in this case. The presence of the large, anterior lying, innominate artery and the position of the superior vena cava on the right side may account for this condition. Wegelin's figure[6] indicates how the goiter is deviated to the left, particularly by the innominate artery.

BIBLIOGRAPHY

1. Higgins, C. C.: Arch. Surg., 15, 895, 1927.
2. Jores, L.: Ziegler's Beiträge z. Path. Anat., 21, 211, 1897.
3. Hesselberg, C.: Frankf. Zeitsch. f. Pathol., 5, 322, 1910.
4. Isenschmid, R.: Frankf. Zeitsch. f. Pathol., 5, 205, 1910.
5. Matti, H.: Schweiz. Med. Wchschnr., 8, 852, 1927.
6. Wegelin, C.: Henke-Lubarsch, Handbuch der Spez. Path., 8, 327, Fig. 104, Berlin, 1926.

CLINIC OF DR. FREDERICK CHRISTOPHER

EVANSTON HOSPITAL

ULCERATIVE ARTERITIS. A CASE REPORT IN WHICH THIS CONDITION OCCURRED AS A COMPLICATION OF ULCERATIVE COLITIS

AFTER a couple of months of relative loss of strength, patient J. B., aged ten years, began to have bloody, watery evacuations in October, 1928. Because of the child's loss of strength, he was sent in December to a Children's Hospital, where, after a period of observation, a diagnosis of ulcerative colitis was made. There were 7 to 9 liquid bloody stools in twenty-four hours and the temperature reached 104 F. The child was put on acriflavine enemata, a special diet, cod liver oil, and opium. The disease followed an up and down course during the first three months of 1929. On March 25th the first injection of a serum for the colitis was given. On March 27th, the second serum injection was given. On March 30th a little furuncle appeared on the leg and by the next day this had become transformed into a small ulcer. An area of inflammation developed on the right arm at the site of the injection. The leg ulcer spread rapidly. Its borders were a dark-blue purple, and its central part was composed of purulent débris. On April 12th the patient was admitted to the Evanston Hospital. At this time there was a systolic murmur at the apex. The ankles were greatly swollen and pitted on pressure. The temperature varied from 98.6 to 102 F. The pulse averaged 110. Urine negative. Hemoglobin 29 per cent; red blood count 2,001,000; white blood count 23,400. A whole blood transfusion of 500 cc. was given by the Scannell method. The patient was then anesthetized and the purple borders of the leg ulcer, which by this time had attained a size of 5 by 6

inches, were excised by the actual cautery. The abscess on the right arm was incised and the necrotic undermined skin removed. An abscess on the right knee was incised and drained. The report of the pathologist, Dr. J. L. Williams, on the excised ulcer border was as follows: "Sections contain skin, subcutaneous tissue, and fat. There is much infiltration with leukocytes, and epithelial cells, and in some places newly formed fibrous tissue. In one place is a pyogenic membrane composed of necrotic ma-

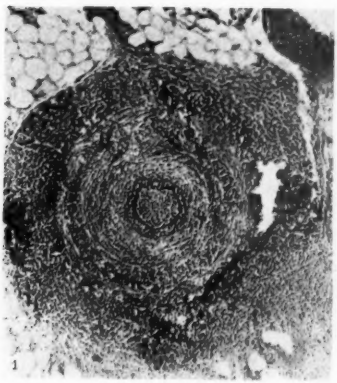

Fig. 125.—Cross section of small artery showing thickening of wall and infiltration with lymphocytes and leukocytes.

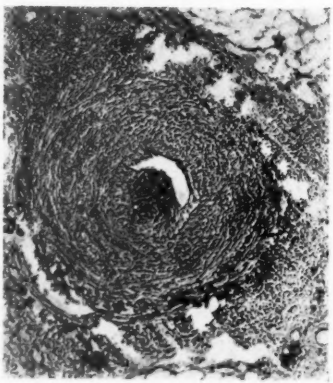

Fig. 126.—Cross section of small artery showing thickening of wall, infiltration with lymphocytes and leukocytes, and partially occluding thrombi.

terial and leukocytes. *A few small arteries are included in which the entire wall is thickened and heavily infiltrated with lymphocytes and leukocytes, and, in a few, partially occluding thrombi are seen* (Figs. 125, 126). Diagnosis: Ulcerative arteritis.* A few gram-negative cocci were seen in the smear, but the plates and anaerobic cultures were sterile. April 15, 1929: Hemoglobin 55 per cent. 4/18/29: Red blood cells, 4,220,000. 4/27/29: 500 cc. whole blood. 4/29/29: Hemoglobin, 68 per cent; red blood

* Words in Italic are by author.

cells, 3,880,000; white blood cells, 11,700. Hemolytic streptococcus isolated from the stool. 5/4/29: Hemoglobin, 77 per cent; red blood cells, 4,230,500; white blood cells, 12,950. 5/7/29: Large ischiorectal abscess incised and drained. 5/11/29: 500 cc. whole blood. 6/5/29: Patient seen by Dr. Clement Debere who made a proctoscopic examination. The distal sigmoid curvature showed the mucous membrane to be very edematous with numerous small ulcerations. A good deal of free blood was present in the rectal ampulla. The ulcers were all small and seemed to be limited to the mucous membrane and not entering the submucous layer. 6/13/29: 500 cc. whole blood. 6/20/29: The surface of the ulcer (which had been given the Carrel-Dakin treatment) was covered with Thiersch skin grafts. These all "took." 6/21/29: Many hyaline casts in the urine, hemoglobin 74 per cent. 7/2/29: Weight, 77¼ pounds. The patient up and about. 7/3/29: Pulse rose from 90 to 130; the average temperature from 99.5 to 101 F. 7/6/29: Dr. Joseph Brennemann, "Pulse rapid, no murmur, heart about 2 cm. out to the left, right border normal. Would keep him absolutely quiet for a few days." 7/12/29: Hemoglobin, 56 per cent; red blood cells, 3,640,000; white blood cells, 7550. Rectal injections of argyrol had been carried out on the orders of Dr. Debere. The patient was discharged from the Evanston Hospital on July 13, 1929. On September 17, 1929, the patient was readmitted for one day for a blood transfusion of 500 cc. of whole blood. After the patient's return home a restricted diet and proper rectal treatments were carried out under Dr. Debere's orders. The patient gradually improved. On November 6, 1929, the patient was beginning to take a few steps, although there was some stiffness of the ankle. The temperature goes a degree or two above normal at night, and the pulse is still elevated. Occasional blood in the stools, but in decreasing quantity. The stools are two or three per twenty-four hours and fairly well shaped. Weight 77 pounds.

The character of the ulcer in this case was of unusual interest. It followed an injection of anticolitis serum but did not appear at the point of the injection. There was no contusion,

abrasion, or wound at the site of the ulcer formation. The ulcer spread very rapidly and its borders were gangrenous. Microscopical sections of the borders showed the vessel walls to be thickened and heavily infiltrated with lymphocytes and leukocytes. The progress of the advancing borders was successfully arrested by excision with the actual cautery.

FRACTURE OF THE SECOND CERVICAL VERTEBRA. A METHOD OF APPLICATION OF A PLASTER CAST TO THE HEAD AND TRUNK

THE taxicab in which Mrs. L. F., aged fifty-eight, was riding was struck by another car, turned over, and set on fire. The patient was extricated without being burned and was brought to the Evanston Hospital on October 13, 1929. Aside from marked pain in the occipital region and abrasions and contusions there were no other symptoms. There were no cord symptoms except possibly inability to completely empty the bladder for the first ten days of her stay in the hospital. The *x*-ray report (Dr. E. L. Jenkinson) follows: "There is a fracture dislocation involving the upper cervical spine. The occiput and first and second cervical vertebrae are dislocated anteriorly and slightly downward upon the third cervical vertebra. There is a fracture involving the lamina of the second cervical vertebra just posterior to the body. The lamina and the posterior spinous process of the second cervical vertebra seems to be in proper relation with the articular surface of the third cervical vertebra (Fig. 127). In the anterior-posterior direction there seems to be very little, if any, lateral displacement of the body."

The patient was placed in bed with sand bags on each side of the head. One to 2 pounds of head traction was obtained by means of a Crile head tractor. By the end of three weeks it was seen that the head traction did not afford sufficient immobilization as the neck became increasingly painful. Accordingly it was decided to place the patient in a head and trunk cast. The application of this cast presented several difficulties. The patient's condition seemed definitely to exclude the application of the cast in the usual vertical position with upward halter traction. The use of the Hawley table would not solve the question, and moreover it was felt that the patient should be subjected to as little movement as possible during the application of the cast.

On November 8, 1929 the cast was applied in the following manner: A strong Balkan frame was placed over the bed and made fast to it. Crossbars were placed over the top of the Balkan frame to give attachment to a number of double block pulleys. Strong canvas bands were placed under the patient's head, axillae, buttocks, and legs, and these bands were attached

Fig. 127.—*x*-Ray showing fracture of second cervical vertebra with forward dislocation of the body of the vertebra. (Outline reinforced for clearness.)

to strong wooden spreaders. Ropes were threaded through the pulley blocks on the spreaders and also through those on the crossbars. With the assistance of an ample number of helpers the patient was gently and slowly raised up in the air to a height of 1 foot above the bed. By proper handling of the head pulley, the desired angle of the neck on the body was obtained. The patient's hands and arms were supported by proper hand

holds. The patient swung free in the air in perfect comfort (Fig. 128). The head, neck, and body, which previously had been enclosed in stockinet, were wound with a thin layer of cotton "sheet-wadding." It is important to have the back of the skull adequately padded. The face was temporarily covered. The plaster cast was then carefully and smoothly applied. When it was completed and had set, the patient was lowered to the bed and the parts of the bands protruding from the cast were cut off. The cast was trimmed away around the face, under the

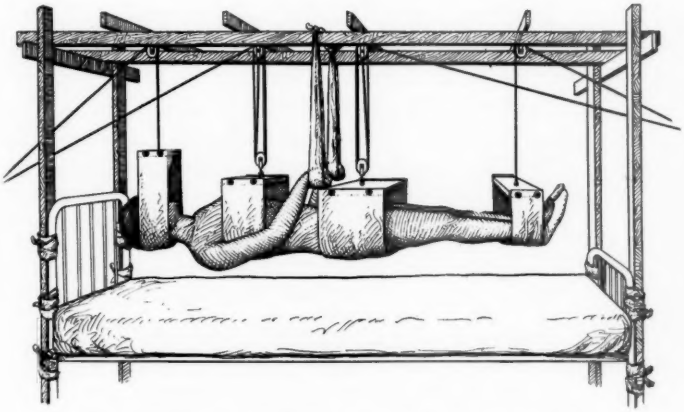

Fig. 128.—Method of suspension for application of a plaster cast in a case of fracture of cervical vertebra. The cast includes the entire head (except the face), the neck, the body to the iliac crests. The arms are left free. The occiput should be well padded to prevent pressure necrosis.

axillae, and over the hips. From time to time further trimming was necessary.

With this support, the neck was held immobile and the patient was encouraged to get up on her feet and take a few steps. At the end of three weeks she had become fairly well adjusted to the vertical position. Accordingly the cast was removed and for it was substituted an adjustable leather and metal neck splint which rested upon the shoulders.

The mortality in high cervical fractures is extremely high.

Death may occur very suddenly. Necessary manipulative procedures may be attended by sudden death. Fractures of the cervical spine without cord symptoms have been reported. A recent one is that of Menegaux.[1] In one of McWhorter's 3 cases of fracture of the atlas it was necessary to remove the posterior arch for neuralgia.[2] Relative immobilization of the neck may be secured by traction, by sand bags, or by the sand-filled leather head rest of Hauflig.[3]

No mention has been found of the application of a plaster cast by the method described above. The nearest approach to it is in the paper of Conwell.[4] This author uses an overhead frame to elevate patients in plaster spica or body casts to facilitate the nursing care. Robinson[5] has described an ingenious table for the application of casts to the neck. It is possible that it might be applied to a case like the present one.

Where there is an anterior dislocation of the atlas there may be an inability to swallow solid foods as was pointed out by Orton.[6]

[1] Menegaux, G.: Rev. d'orthop, 14, 229.
[2] McWhorter, G. L.: Jour. Bone and Joint Surgery, 11, 286, April, 1929.
[3] Hauflig, S. S.: Jour. of Bone and Joint Surgery, 11, 33, January, 1929.
[4] Conwell, H. E.: Surg., Gynec., and Obst., 46, 576, April, 1928.
[5] Robinson, W. H.: Amer. Jour. of Surg., 2, 595, June, 1927.
[6] Orton, H. B.: Laryngoscope, 36, 188, March, 1926.

NECROSIS OF ILEUM FOLLOWING PELVIC INFLAMMATORY DISEASE

MRS. E. J. S., aged thirty-one, was admitted to the Evanston Hospital on October 31, 1929. For the last few months she had been troubled by fatigue, indifferent appetite, dizziness, and constipation. She had had no pregnancies or miscarriages and her last menstruation had been two weeks previously. The patient began to have severe abdominal pain at 4 A. M. on the day of her admission to the hospital. This pain seemed to originate in the region above the symphysis and spread over the entire abdomen. On admission to the hospital at about 10 A. M., examination of the heart and lungs gave normal findings. The right lower abdomen seemed to be slightly more rigid than the left. There was considerable tenderness, the maximum point of which was possibly a little to the left of the midline. The pelvic examination was indefinite. The urine was negative, the temperature 98. F., pulse 100, respirations 30, but the leukocyte count was 27,850. A tentative diagnosis of acute appendicitis was made and operation was determined upon.

In view of the fact that the diagnosis was not perfectly clear the patient was placed in Trendelenburg position. On opening the peritoneum through a midline incision, a small quantity of blood-stained serous fluid was found. The appendix was found with some difficulty and upon inspecting it, it was found to be bright red and injected, but not having the typical characteristics of acute appendicitis, but rather those of periappendicitis. The appendix was removed by ligation and linen purse-string inversion. The pelvis was then explored and the uterus and tubes were found to be bound down by dense adhesions. There was also bound down in the pelvis, a loop of intestine which was densely adherent to the top of the uterus. The loop of intestine was freed with some difficulty and was found to be a dark purple color for a distance of some 8 inches. The

tubes were then freed and the uterus was drawn forward by a tenaculum. Both tubes were found to be markedly diseased and were tortuous and injected. On the side of the uterus in the left broad ligament was a large, white calcified nodule. There were white patches on both ovaries. Both tubes were removed, but the ovaries were allowed to remain in an effort to preserve their blood supply. The purple loop of bowel beforementioned was now inspected, after having been left in the abdominal cavity. It was found still to be purple. It failed to respond to pinching and other stimuli, to give it peristolic movements. It was placed upon the abdominal wall and covered with hot, moist towels for a period of some ten minutes. This treatment, however, failed to restore its apparent viability, and it was thought necessary to do a resection. Accordingly, some 10 to 12 inches of the ileum at a distance of about 1½ feet from the ileocecal valve, was resected and a lateral anastomosis was carried out. The butt ends of the cut-off ileum were inverted with linen suture. The anastomosis was carried out in three layers with No. 1 chromic catgut or fused needles. The aperture in the mesentery was proximated by interrupted catgut. The patient's condition throughout was satisfactory. The peritoneum was closed with plain catgut; the aponeurosis was closed with chromic catgut; two stay sutures over buttons were put in place; clips used for the skin.

The report of the pathologist, Dr. W. W. Brandes, is as follows:

"An appendix measuring 5.5. cm. in length, and 7 mm. in diameter. The surface of the distal half shows what appears to be tags of adhesions that are diffusely reddened. The lumen contains a small amount of reddish-gray exudate. It is obliterated in the distal third. The mucosa shows numerous ulcerations surrounded by hemorrhage.

"One of the tubes measures 8 cm. in length, diffusely reddened. The broad ligament attached to it is also markedly injected. The lumen is patent the entire length. The fimbriae are swollen and injected. The mucosa is reddened. A portion of the other tube measures 5 cm. in length. The fimbriated end is closed by

adhesion. The tube is slightly dilated in the distal portion. To the fimbriated end is attached a hard white smooth mass, 7 by 12 mm., a part of the uterine wall. The lumen of the remainder of the tube is patent. The mucosa is markedly swollen and edematous. A piece of small bowel measuring 24 cm. in length is diffusely reddened for 22 cm. of this extent and small areas show dark blue to almost black discoloration. The wall is thickened and edematous. The mucosa is purple in color and edematous in appearance.

"Sections: The wall of the tube is thickened. Numerous dense cells, mostly lymphocytes, large round cells, less numerous plasma-like cells, and a few polymorphs. The papillae in the lumen are long, some thickened and cellular.

"The appendix shows hemorrhages and areas of round-cell infiltration in the outer portion of the muscular and the serosal layers.

"The mesenteric vessels do not show thrombi; some areas of hemorrhage are present.

"*Diagnosis.*—Chronic appendicitis and salpingitis. Necrosis of bowel with marked edema."

On the second postoperative day the patient developed a dilatation of the stomach which quickly cleared up after repeated lavage, withholding of mouth fluids, and the administration of fluids by rectum, infusion, and under the skin. The convalescence was otherwise uneventful. A vaginal smear for gonococci was negative. The patient was discharged from the hospital on November 16, 1929 and has gained strength rapidly since she has been home. She says that she has felt better than she had felt in a long time.

REPAIR OF HEPATIC DUCT

MRS. S. M., aged twenty-four, who had in recent years been very obese, but who now was of average weight, was delivered of a baby in March, 1929. During April, May, and June she had had several attacks of gallstone colic. On June 27, 1929 a cholecystectomy and an appendectomy were done. The gallbladder, which contained numerous stones, was non-adherent and readily isolated. It was removed from below upward. The cystic duct was dissected out and clearly defined before section. A second structure, which was deemed to be the cystic artery, was also ligated and sectioned. In the report of the pathologist, Dr. J. L. Williams, was included the following: "About 2.5 cm. of the cystic duct is included with the specimen and near the end of this is about 1 cm. of bile duct lining which may be a part of the cystic duct partially severed from the remainder at the time of operation." After the operation the patient became deeply jaundiced, the stools were clay colored, and bile appeared in the urine. On July 10, 1929 an exploratory operation was carried out. The region of the bile ducts was carefully exposed and it was discovered that some $1\frac{1}{2}$ to 2 cm. of the hepatic duct just proximal to its attachment to the cystic duct had been removed at the previous operation. A small stub of the cystic and hepatic ducts remained before they merged into the common duct. These stub ends were split open so as to make a common lumen. The proximal cut end of the hepatic duct was about even with the lower border of the liver. There was so much loss of substance that the cut ends could not be approximated, and the proximal end was so short that an anastomosis to the duodenum could not be carried out. Accordingly it was decided to attempt the repair by the "anchored tube" method of L. L. McArthur. In this method a rubber catheter is placed into the common duct down into the duodenum for a distance of several inches and

the proximal end is inserted into the proximal hepatic or common duct. The tube is anchored to the surface skin by a strong silk ligature until such time when the epithelization of the bile duct is thought to be complete and then it is cut loose and tug of the duodenum draws the catheter and ligature down into the duodenum and they are passed by rectum. In the patient at hand it was found impossible, however, to force the smallest catheter through the ampulla of Vater, until a strong

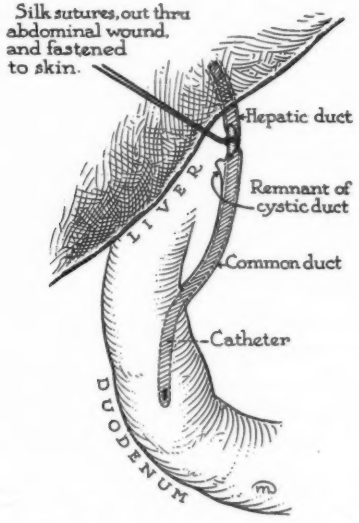

Fig. 129.—Injury of hepatic duct with loss of substance. Diagram showing method of repair.

probe had been placed inside of the catheter to stiffen it. Even then it was forced into the duodenum for a distance of some 2 inches only with great difficulty. The proximal end of the catheter was inserted into the proximal end of the hepatic duct for a distance of about 1 inch. The ragged ends of the bile duct were approximated as well as possible. A strong braided silk ligature was then placed about the catheter and brought up through the abdominal wound and was made fast to the surface

skin by adhesive (Fig. 129). Three cigaret drains were used. The patient's condition after the operation was very poor. On the first day the pulse was as high as 168 to 170. On the eleventh postoperative day she developed a dilatation of the stomach which was remedied by lavage. After a stormy convalescence she finally was discharged from the hospital on August 11th. At this time she was discharging great quantities of bile through the fistula which contained the silk ligature. The stool continued to be clay colored, but the jaundice disappeared. The patient was fed oxbile in capsules, but they were not tolerated by the stomach. Bile salts seemed to have no beneficial effect. The stools, which had been hard and dry while in the hospital, became watery and frequent soon after the patient returned to her home. Dietetic precautions and bismuth were unable to influence the diarrhea, which had come to cause the patient more suffering than the biliary fistula. Despite a liberal diet the patient continued to lose weight and was very miserable. On September 21, 1929, the seventy-third day after the second operation, the patient's weight was $96\frac{3}{4}$ pounds. On this date careful traction was made on the silk ligature and the entire rubber catheter was withdrawn. The distal 2 inches of which had protruded into the duodenum was found to be covered with a firm black deposit. On the following day the patient called up with the startling information, "I am all right now, doctor. There is no bile coming out of the fistula, the diarrhea has stopped, and the stools are brown." And save for a small discharge of bile from the wound about a week later, this improvement was maintained. The patient gained over 10 pounds in six weeks and naturally her spirits improved tremendously. She resumed the care of her baby and left for another city to take up her stenographic work.

In this connection Muzeneek,[1] after 53 experiments on dogs, came to regard the bridging of the defect with a rubber drain, covered with suitable tissue flaps, as the most practical method of plastic repair of fresh injuries of the bile ducts. He

[1] Muzeneek, P.: Deutsche Ztschr. f. Chir., 195, 267, abstracted Jour. Amer. Med. Assoc., 86, 2005, June 26, 1926.

found omentum and segments of veins to be the best covering. He believed that the drain should be left in as long as possible. McWhorter[1] has found that after operations on the common duct diversion of the bile is an aid to primary union. Where there is little or no loss of substance this author believes that the overlapping method of anastomosis with the "riband" common duct gives a larger lumen and a firmer union than the end-to-end method.

Hopes for a complete recovery after this operation began to wane when in December the patient began to have itching of the skin and a highly colored urine. During January the stools became clay colored, a troublesome diarrhea existed, and the skin became deeply jaundiced. The patient returned home and a diagnosis of cicatricial stenosis of the bile duct was made. The patient was readmitted to the Evanston Hospital on February 5, 1930, and two days later, with the assistance and counsel of Dr. L. L. McArthur, a second plastic operation on the bile duct was carried out. After separation of the adhesions and careful sharp dissection of the dense scar tissue at the site of the previous common and hepatic ducts, the slightly dilated stub end of the common hepatic bile duct was found at the liver border just where it originated from the union of the right and left hepatic ducts. A small section of discolored mucosa buried in scar tissue about midway between the liver and duodenum was all that was found of the common duct. A silk purse string suture was placed in the duodenum in the portion nearest the liver. In the center of this purse string a small incision was made in the duodenum and a small rubber catheter (about 16 F.) in which an extra eye had been cut was passed for its entire length, save the last 2 to 3 cm., through the hole into the duodenum. The purse string was drawn up snug and a second purse string was put in place. The duodenum was then readily brought over to the liver border. The proximal portion of the rubber catheter was split into two halves and the ends tapered. These ends were passed up into the right and left hepatic ducts. The mucosa of the hepatic duct was sutured around the tube and also to the

[1] McWhorter, G. L.: Arch. Surg., 18, 117, January, 1929.

duodenum, using silk, so that no space intervened between the hepatic duct and the duodenum. The wound was closed with drainage. The patient has made a rapid and uneventful recovery. Fifteen days after operation she walked into the office feeling very well. Her skin was white and the stools were bile stained. The diarrhea had disappeared and she had already gained some weight.

BENIGN OBSTRUCTION OF THE SIGMOID

PATIENT G. P., aged thirty-eight, was admitted to the Evanston Hospital on September 25, 1929. His chief complaints were pain in the lower left quadrant and irregularity of the bowels. For the past two months he had been troubled with constipation, often going three days without a bowel movement. There had been no diarrhea nor blood in the stools. For three weeks there had been discomfort in the left lower quadrant which had developed into actual pain for the last three or four days. There had been no previous illnesses. On admission the temperature, pulse, and respirations were normal. The Wassermann in the blood was negative. The urine was negative. The leukocyte count was 12,000, with 78 per cent polymorphonuclears. A hard tender mass was palpated in the left lower quadrant. The *x*-ray report (Dr. James T. Case) on September 26, 1929 is as follows: "Under the fluoroscope it was seen that the enema entered the colon and progressed upward with fair ease. There was noted a filling defect in the colonic shadow just below the crest of the left ilium. This was coincidental with a palpable hardness which felt like a lump through the abdominal wall. The enema passed this filling defect without hindrance. The colon filled throughout to the cecum with ease. A carcinoma in this region or an organic intrinsic obstruction is usually accompanied by considerable difficulty in passing the opaque fluid beyond the narrowing. The lesion is constant in all the films. There is apparently an organic lesion in the descending colon just below the crest of the left ilium seen fluoroscopically and on repeated films. Spasm really seems an inadequate explanation. . . ." After systematic administration of atropine, the report continues, "Twenty-four hours of administration of antispasmodics shows the same filling defect with essentially the same detail characteristics" (Fig. 130).

The patient was readmitted on September 30, 1929 and operation was done on October 1, 1929.

The patient was placed in a Trendelenburg position and a long left paramedian incision was made, extending from above the symphysis to well above the umbilicus. On opening the peritoneum, the upper sigmoid flexor, and descending colon were found to be densely adherent to the lateral parietal wall.

Fig. 130.—*x*-Ray showing filling defect in barium-filled sigmoid. A benign obstruction.

These adhesions were carefully separated by the finger. An abscess containing about 1 ounce of creamy yellow pus was broken into at this point (a subsequent culture of this pus showed staphylococcus) with great care. The adherent mass was further dissected free and the peritoneal reflexion was at this point turned mesially. In this manner the hard, inflammatory mass, which at one point had a puckered appearance and resembled carcinoma, could be turned to the midline. The

vessels of the mesentery which supplied this part were ligated and the affected section of the bowel with about 2 inches above and below was resected. The cut ends were cauterized and turned in with a double layer of chromicized catgut. A lateral anastomosis about $2\frac{1}{2}$ inches in length was then made, using linen for the inner layer and chromic catgut for the outer layer. There was no evidence of damage to the ureter. The appendix presented readily and was quickly removed by ligation and linen purse-string inversion. The wound was closed by plain catgut for the peritoneum, chromic catgut for the fascia, with three or four stay sutures with buttons.

The report of the pathologist, Dr. W. W. Brandes, is as follows:

"This specimen consists of fat, a portion of the large bowel 12 cm. in length. The fat surrounding this is markedly indurated. The cut surface shows irregular areas of yellow fat surrounded by firm grayish areas. The mucosal surface in the region of this induration surrounding the tissue is reddened, but shows no ulceration. There is one soft dark-red degenerated area in the surrounding fat.

"An appendix 8 cm. in length and 6 mm. in average diameter. The vessels of the surface are slightly prominent. The lumen contains soft fecal matter. The wall is slightly thickened and gray. The mucosa is uniform gray.

"Sections of the mass show a low-grade or subacute inflammatory mass. The mucosa appears to be intact and the mass is made up largely of very dense fibrous tissue with infiltrated cells, polymorphs, eosinophils, plasma cells, and lymphocytes. The fat in mesentery is also richly infiltrated with cells more rich in polymorphs.

"The appendix shows no unusual change.

"*Diagnosis.*—Subacute nonspecific inflammatory mass."

The convalescence was extremely stormy. Dilation of the stomach responded to repeated washings of the stomach. Nearly the entire laparotomy wound became infected and broke down. It rapidly improved under Carrell-Dakin treatment. Frequent hypodermoclyses and infusions were given. On October 8, 1929

the urine showed albumin ++, sugar +++, and many granular casts. The next day the leukocyte count was 16,350. On October 13, 1929 a fecal fistula developed from the stab drainage wound in the left flank. On November 28, 1929 the fecal fistula was almost closed; there was a slightly tender mass in the lower left quadrant; and the temperature averaged about 99.5 F. The urine was negative. On December 4th, following increasing tenderness and temperature, an abscess was opened in the left lower quadrant, and a few days later a small incisional abscess opened spontaneously. The patient has been given daily enemata, which have been successful for feces and flatus. He has been eating well, and his temperature is normal. On February 1, 1930 his wounds were entirely healed and he was out-of-doors taking walks

It is possible that in this case it might have been a safer procedure to have done the operation in two stages. At the first operation merely to have drained the abscess, and later to have done the resection. However, there was no complete assurance that the infection could have been entirely cleared up by such a procedure.

ILEOCOLIC INTUSSUSCEPTION WITH MECKEL'S DIVERTICULUM

BABY JOHN H., Jr., was an exceptionally well nourished and robust infant of nine and one half months. Save for the passage of a little blood per anum in the early days of its life its history was negative. On the morning of November 18, 1929 the mother observed that the child was somewhat listless and called a physician. The latter was unable to discover anything out of the way except a slight respiratory infection. During the day the child vomited a number of times and seemed occasionally to be in transitory pain. At 7.30 P. M. the same day the patient was seen by Dr. Joseph Brennemann. At this time there was a palpable tumor in the right lower quadrant and blood had been passed by rectum. Dr. Brennemann immediately made a diagnosis of intussusception and the child was operated upon at the Evanston Hospital about one and one half hours later.

A right paramedian incision was made and the rectus muscle was retracted mesially. A small quantity of clear, straw-colored fluid escaped on opening the peritoneum. After retracting the intestine, a matted piece of bowel was found in the ileocecal region. The cecum was identified and was found to contain a doughy mass. The ileum, adjacent to the cecum, was affected for a distance of some 10 or 12 inches. The ileum in this region showed tortuous coils which were of a purplish color. They appeared to pass down underneath through a hernial opening, or cord, so that the appearance of a volvulus was presented (Fig. 131). This, however, was not the case. By careful examination, the entrance of the intussusception was found. After considerable difficulty in manipulation the cecum was compressed and squeezed so that the telescoped end began to enter the ileum. The ileum was then massaged and graudally the telescoped intestine was opened up. When it was completely

reduced, it was found that the starting point of the intussusception was a Meckel diverticulum. The bowel of the intussusception was found to be dark-red for a distance of some 8 inches. The entire circumference of the bowel was not uniformly affected. After putting hot towels on the affected bowel for a period of time, the color was thought to improve slightly. The serosa was shiny throughout. The patient's condition at

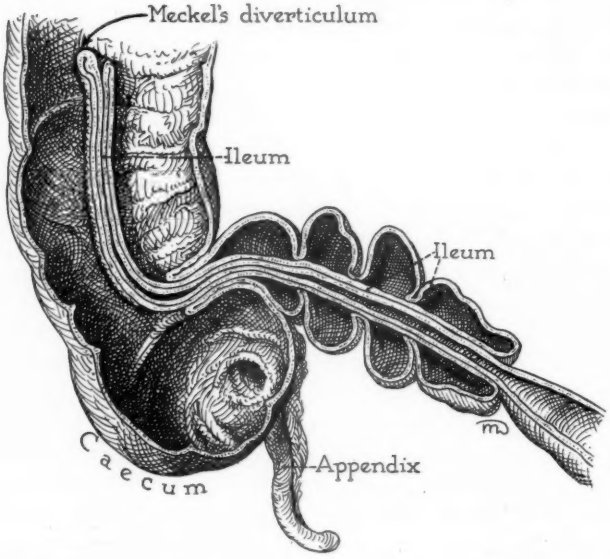

Fig. 131.—Diagrammatic representation of condition found at operation. An ileocolic intussusception initiated by a Meckel diverticulum.

this time was very poor, the pulse being in the neighborhood of 190. While a resection of the affected intestine seemed to be indicated, it was felt certain that the baby would not survive such a procedure. In view of the fact that there was some encouragement in the return of color to the bowel, no further procedure was taken and no effort was made to deal with the Meckel diverticulum. The peritoneum was closed with plain catgut, the fascia with No. 1 chromic catgut, and silk for skin.

A hypodermoclysis was given in the thigh of normal saline. Patient was left on the operating table for an hour with hot-water bottles and the head held down, after which time the condition improved considerably.

After the child's return to his bed his condition improved rapidly. The following morning his general appearance was excellent. In the afternoon, however, the abdomen became more and more distended and the temperature rose to 105 F. rectal. The child had a few mild convulsions and vomited. The hopes that the affected loop of bowel was viable or that it would not interfere with the passage of feces and flatus began to wane. Accordingly at 11 P. M. on November 19, 1929 the second operation was undertaken.

The old incision was opened up without anesthesia, and the dark loop was readily found and delivered on the surface of the abdomen. The abdomen was closed with interrupted master sutures through and through, involving the peritoneum, fascia, muscle, and skin. Braided silk was used. Abdomen was then closed, all but a small opening, through which the purple loop of gut had been withdrawn. This was carefully walled off with vaselin gauze, and owing to its distended condition, and the poor condition of the patient who had been having a convulsion on the operating table, as well as before, the gut was opened and a large catheter was passed down into the lumen of the proximal segment through into the abdomen, through into the intra-abdominal portion of the ileum. Immediately a large quantity of gas escaped and the abdomen became softer and the patient breathed more easily. A similar catheter was passed into the distal loop so as to enter the cecum. Patient's condition at the end of the operation was better than at the first.

The child's improvement, however, was but temporary and after rapidly recurring convulsions died at 4 A. M. on November 20, 1929. A postmortem examination was done in addition to the pathology already described, the only other noteworthy finding was enlarged thymus weighing 32 Gm.

LATERAL CERVICAL FISTULA. PARAFFIN INJECTION AS AN AID TO EXCISION

PATIENT R. R., aged thirty-two, was admitted to the Evanston Hospital on October 23, 1929. He was in the care of Dr. John B. McClellan for a bleeding duodenal ulcer. After three or four days the stools were free of blood and Dr. McClellan consented to an operation which the patient had requested for a lateral cervical fistula. Accordingly, under ethylene anesthesia, operation was done on October 28, 1929. By pressure upon the sinus several large drops of creamy yellow pus were milked out. By means of a blunt needle, the sinus was first injected with methylene blue. For some years Dr. S. W. McArthur has advised the injection of such sinuses with paraffin. The paraffin was melted in a sterile basin placed in the steam sterilizer. A 10-cc. Luer syringe with a blunt silver needle was placed in a basin of hot water. After the hot syringe had been filled with the molten paraffin, the needle was quickly attached and placed into the orifice of the fistula. It was found, however, that the silver needle cooled too quickly and that the paraffin solidified in its lumen. By wrapping the needle in gauze moistened in very hot water (Fig. 132) the paraffin could very quickly and easily be injected into the fistula. When no more paraffin could be injected, it was felt to have hardened, and the fistula could easily be palpated in the neck as a hard cylinder. A longitudinal incision was then made over this mass and due to its hardness and the fact that it was colored blue it was easily dissected out. Its proximal portion ended blindly behind and above the hyoid bone and did not extend into the pharynx. A split rubber tube drain was put in place and the skin wound was closed with Michel clips. A short time after the patient's return to his room from the operating room his respirations seemed to have stopped entirely, although the pulse

remained strong. After artificial respiration and lobelin had been administered, the breathing was resumed and the patient went on to a rapid and uneventful convalescence.

In a previous paper on lateral cervical fistula[1] the writer subscribed to the work of Wenglowski[2] which showed these

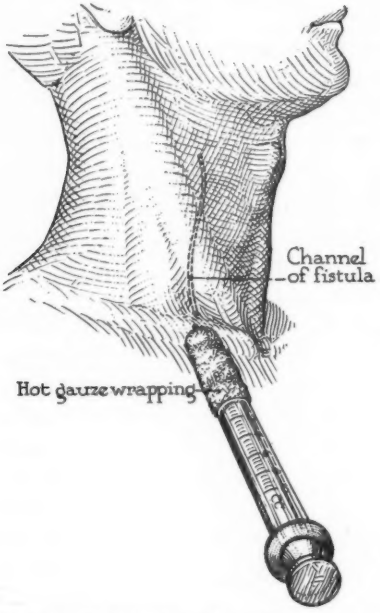

Fig. 132.—Method of paraffin injection in lateral cervical fistula. Note needle wrapped in hot moist gauze.

fistula to be remnants of the embryonic thymopharyngeal duct and not of the branchial apparatus. This theory is also supported by Gross,[3] but is disputed by Mueller[4] and Nylander.[5] The *x*-ray examination of these fistulæ has been aided by injec-

[1] Christopher, Frederick: Surg., Gynec., and Obst., March, 1924, p. 329.
[2] Wenglowski, R.: Arch. f. klin. Chir., 1912, 789.
[3] Gross, W.: Zentralbl. f. Chir., 53, 2076, August 14, 1926.
[4] Mueller, S.: Deutsche Ztschr. f. Chir., 193, 401, 1925.
[5] Nylander, P. E. A.: Deutsche Ztschr. f. Chir., 215, 139, 1929.

tions of lipiodol[1] and of bismuth and oil.[2] In cases of complete fistula Wooden and Hutchens[3] have noted that after mastication of a methylene blue tablet, there was prompt external appearance of the color. Carp[4] called attention to the fact that the vagus is so situated that it is most likely to come into contact with a "branchial" fistula. It is possible that this latter fact may have some relation to the respiratory difficulty experienced not infrequently in these cases.

[1] Comer, M. C.: Southwestern Medicine, 11, 308, July, 1927.

[2] Meyer, H. W.: Amer. Jour. of Surg., 40, 121, May, 1926; Kramer, R.: Laryngoscope, 36, 517, July, 1926.

[3] Wooden, W., and Hutchens, D. K.: Amer. Jour. of Surg., 3, 377, Octover, 1927.

[4] Carp, L.: Surg., Gynec., and Obst., 42, 772, June, 1926.

CLINIC OF DR. BERNARD PORTIS

MICHAEL REESE HOSPITAL

A CASE OF CORONARY THROMBOSIS SIMULATING AN ACUTE SURGICAL CONDITION OF THE ABDOMEN

MANY cases have been reported of coronary lesions simulating acute surgical conditions of the abdomen. The differential diagnosis of these two lesions is frequently confused. This case is reported to demonstrate many of the significant features and the invaluable aid of the electrocardiogram.

Mr. A. H. S., a patient of Dr. Milton Portis for many years, consulted us on October 12, 1929 because of very severe, cramp-like epigastric pain. This pain radiated to the back and down the arm. It was associated with excessive belching, but there was no nausea or vomiting. The physical findings revealed a man of middle age, well developed, and suffering from severe abdominal pain sufficient to double him up and complain bitterly of his abdominal symptoms. On further examination no local tenderness or rigidity could be elicited in the abdomen. The temperature was normal and the white blood count was 8000. The probable diagnosis of a coronary lesion was seriously considered. He was given $\frac{1}{8}$ grain of morphine and put to bed.

The patient had several attacks the same day of a similar character. The leukocyte count remained normal. The blood pressure at one time during the attack rose to 150 systolic and 98 diastolic. His previous blood pressure ranged about 130 systolic and 80 diastolic. An electrocardiogram (Fig. 133) was taken during the attack, which showed several features which were of great significance, namely, a tachycardia of 100 and very atypical configuration of the T wave. In the first lead of the electrocardiogram the T wave was diphasic and in the second

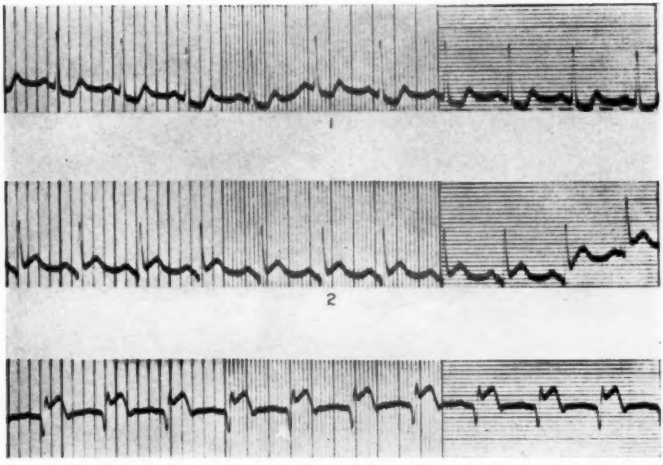

Fig. 133.

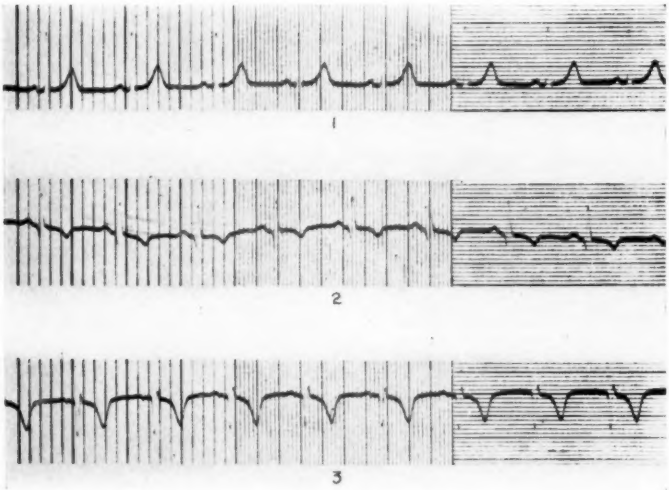

Fig. 134.

and third leads the T wave started above the base line without the usual S–T interval.

About three days later another electrocardiogram (Fig. 134) was taken which very classically demonstrated the presence of a coronary lesion with inversion of the T wave in lead II, and an extreme negativity of this wave in lead III.

This patient has been observed at frequent intervals. His condition has outwardly improved considerably, so that he is now able to assumed part of his usual activities. The last electrocardiogram (Fig. 135) taken on December 2, 1929 still showed

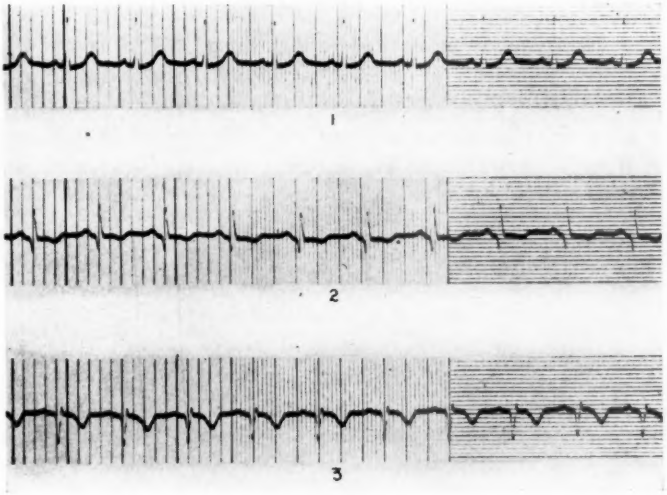

Fig. 135.

findings similar to the previous one with the exception of a lessened negative direction of the T wave in leads II and III.

Summary.—This case clearly demonstrates the fact that coronary thrombosis may at times present the picture of acute surgical condition of the abdomen, and it is very important for the surgeon to recognize this possibility and to utilize the electrocardiogram in aiding the differential diagnosis in the atypical upper abdominal catastrophes, more especially the various colics, ruptured peptic ulcers, diaphragmatic pleurisy, gastric crises, and acute pancreatitis. The physical findings in this case are

conspicuous by their absence, and not consistent with the severity of the pain. The laboratory findings in this patient indicated an absence of an inflammatory condition with a normal leukocyte count, an icterus index of only seven.

Electrocardiographic studies are now easily available and should be used with greater frequency. The portable apparatus may be readily taken into the home of the patient if the condition demands it. The early significant electrocardiographic findings of coronary thrombosis are indicated by abnormal changes in the ventricular complex, especially variations in the T wave which later takes a negative direction. The Q. R. S. complex is frequently widened and notched, especially if the occlusion of a coronary vessel leads to changes in the bundle branches.

This case further emphasizes the necessity of cooperation between surgeons and the internist in atypical surgical abdominal conditions.

REPORT OF FIVE CASES OF FULMINATING APPENDICITIS IN CHILDREN WITH A LOW LEUKOCYTE COUNT

ACUTE appendicitis in children has received considerable attention for many years, yet a careful analysis of a group of cases frequently brings new facts to light and gives a personal reaction to some of the accepted tendencies and ideas. These 5 cases, collected from the author's series of 100 consecutive children with acute appendicitis, demand special consideration because of the fulminating type of the condition and the presence of a low leukocyte count. These patients were operated on from the Children's Ward of the Michael Reese Hospital and the author's private service. They all demonstrated certain features which led to the early diagnosis of the condition and I should like at this time to mention some of the symptoms on which I place great significance.

First, the pain is a variable factor in children and the evaluation of the severity of this symptom requires careful observation. In general, appendicitis rarely starts in with severe abdominal pain except in those cases in which the appendix is retrocecal in position and accompanied with irritation of the underlying ureter, when one may expect symptoms simulating ureteral colic with the presence of blood in the urine. These 5 cases here reported showed in 4 patients at first generalized and epigastric pain and only after several hours did it localize in the right lower quadrant. The fifth patient started with right lower quadrant pain at once.

Second, gastro-intestinal distress was present in all cases. In two the earliest manifestation was that of simple anorexia or loss of appetite. The other three had distinct nausea and vomiting within six hours after the onset. I have never seen any case of appendicitis in which some form of gastro-intestinal distress was absent.

Third, the temperature ordinarily is increased in the first twenty-four hours. However, very characteristically in the children under consideration the temperature rose rapidly to 103 or 104 F. within twelve hours after the onset. The pulse was somewhat increased, ranging around 120.

Fourth, the physical findings are very important, especially that of localized tenderness in the right lower quadrant. The degree depends somewhat on the proximity of the appendix to the anterior abdominal wall. Exquisite rebound tenderness, which may be elicited by suddenly letting up the compressing hand, is usually diagnostic of peritoneal inflammation or free peritoneal exudate. Several German authors have noted the absence of the superficial abdominal reflexes in the right lower quadrant in the presence of acute inflammatory conditions of the appendix. Muscular rigidity is the most difficult to evaluate of all physical findings. In the early stages it is frequently impossible to note its existence, while later with more extensive peritoneal involvement rigidity is constant. Rectal examination should always be done and is usually quite helpful in low-lying and retrocecal appendices. In girls one should be cautious not to confuse the cervix uteri for the appendix.

Fifth, the leukocyte count is usually increased, the majority of my cases of the larger series ranging between 15,000 and 20,000 and a definite polynucleosis. The white counts in the 5 cases being considered ranged from 4800 to 7200.

These five patients were operated on shortly after being seen and all demonstrated a gangrenous condition of the appendix. All of these patients made an uneventful recovery.

In the diagnosis of acute appendicitis in children, even after a very careful analysis of the history, symptomatology and physical findings, there are numerous obstacles in arriving at a correct diagnosis. In many of the borderline cases the diagnosis is made only after the exclusion of other conditions, namely, acute pyelitis, gastro-enteritis, upper respiratory tract infections, mesenteric lymphadenitis, intussusception, acute Meckel's diverticulitis, pneumococcic peritonitis, spondylitis, osteomyelitis of the neck of the femur, cyclic vomiting, cecal tuberculosis, and

any of the numerous causes of acute surgical abdominal conditions which occur in adults.

Treatment.—I believe in the dictum that once the diagnosis of acute appendicitis is made, operation should be performed without delay and that the appendix should be removed in all cases where it is humanly possible without subjecting the patient to too great a risk. I will not consider the removal of the ordinary acute appendix in which there are no peritoneal complications. I fully realize the diversity of opinion as to the advisability of removing the appendix in the presence of an abscess or peritonitis. The appendix was removed in all of the series of 100 cases except one seventeen-month-old baby seen late with an appendiceal abscess located in the region of the gallbladder. I do not believe, with the patient in the proper hands, with careful technic, light anesthesia, that the risk is increased by removing all appendices. In some cases of extensive gangrenous and perforated appendicitis very little may remain of the previous appendix to remove, but I do advocate the ligation of the appendiceal stump and its invagination into the cecum. This prevents further contamination of the peritoneal cavity with fecal material and the future development of secondary abscesses and fecal fistulae. I personally have never had a fecal fistula occur in children or adults following this technic.

There are several features which I should like to mention in the postoperative care of these severe complications of acute appendicitis. Any patient with peritoneal involvement should be considered as a potential case of paralytic ileus if this latter condition is not already present. Because of this all such patients are put up in Fowler's position, no fluids are given by mouth or rectum, hypodermoclysis alone is the route of choice for supplying the body fluids until one is certain that the peristalsis has reestablished itself, which may require between two and four days. No stimulating enemas are administered during this period. Also, I believe, the peristaltic stimulants are definitely contraindicated until the intestinal musculature has regained its normal tone. Any vomiting should immediately be

followed by frequent and repeated gastric lavage. Ordinarily the patient will start to pass flatus about the third or fourth day and then, if necessary, small doses of pituitrin may be administered. Blood transfusion may be utilized in very severe cases. The feeding is gradually increased, starting with ice pellets, small quantities of water, and orange juice. If this is well tolerated the diet should be slowly and carefully increased. Sedatives should be administered with great caution in children. The high rectal tube should be inserted at regular intervals during the stage of paralytic ileus and until the distention has completely disappeared.

Summary.—These 5 cases taken from a larger series illustrate the occurrence of fulminating appendicitis in children. The symptoms which demand special emphasis are the early diffuse moderate pain, the constant presence of gastro-intestinal disturbances, the rapidly increasing temperature, the low leukocyte count, the tenderness in the right lower quadrant, and the rapidity of the process leading to early gangrene.

CLINIC OF DR. RALPH BOERNE BETTMAN

FROM THE SURGICAL SERVICE, MICHAEL REESE HOSPITAL

CLINIC ON MECKEL'S DIVERTICULUM

VOLVULUS OF A MECKEL'S DIVERTICULUM

THE case I wish to present is of interest because a Meckel's diverticulum was the seat of the trouble and because the pathologic condition consisted of a volvulus of the diverticulum itself with a consequent strangulation and gangrene.

The patient walked into my office complaining of abdominal pain with a slight degree of nausea. Four years ago he had been sick with what had been diagnosed as an attack of acute appendicitis from which he speedily recovered without operation. He had been perfectly well since then. Four or five days ago he had had mild abdominal pains, "like gas pains," which had lasted for an hour or so and which had been relieved by a bowel movement. The pain did not return and he was able to carry on his usual business routine until this morning. He wakened this morning with a slight abdominal pain and no appetite for breakfast. During the morning the pain increased in severity. At times the pain seemed to be referred to the region of the urinary bladder, and at such times he would have a desire to urinate. The commencing of urination seemed to aggravate the pain. There was no blood in the urine as far as he could tell. As the morning wore on the pain became more definitely localized to the umbilical region and to the lower right abdomen. He did not vomit. He had no chills.

On examination I found a man of about thirty-five years old, who did not look very sick, and who walked erectly, although complaining of abdominal pain. No abnormalities were noted except for the abdominal findings. The abdomen was slightly

distended, there was a definite although very slight involuntary muscle resistance. This was most marked over the lower right abdominal quadrant. On deeper palpation exquisite tenderness was elicited over the entire lower right abdominal quadrant. There was a definite muscle defense reaction over the right abdomen, especially over the lower part. Over umbilical region and lower left abdomen, muscle resistance was also present, but much less marked than over the right side. There was no lumbar rigidity. Rectal examination revealed no abnormalities. Urine was normal, temperature 101.4 F., and his leukocyte count was 17,200. The diagnosis of acute appendicitis was made and I sent him to the hospital for operation.

I reconciled the urinary symptoms with my diagnosis by postulating either a deep lying appendix or a retrocecal appendix. This would have fitted in very well with the absence of involuntary muscle spasm and with the absence of pain on light palpation, but with the exquisite tenderness and muscle spasm on deep palpation. An inflamed appendix lying over the course of the ureter can produce almost all the symptoms of ureteral calculus including hematuria. I have seen cases of retrocecal appendicitis in which hematuria was one of the first symptoms, and not so many years ago one of my colleagues was nearly operated upon in a nearby clinic for a supposed nephrolithiasis and pyelitis when what he actually had was an inflamed retrocecal appendix with abscess formation. As a rule such cases have defense spasm of the lumbar muscles, but that is not always the case.

The patient was operated upon about three hours after I had first seen him or about nine hours after the beginning of his attack. On opening the peritoneal cavity a few cubic centimeters of clear serous fluid escaped. The cecum which presented into the wound was slightly injected. The appendix was longer than normal, thicker than normal, and the serosa injected. A firm band of adhesions extended from the cecum to the midportion of the appendix, binding it to the cecum at this point. The appendix might very well have been the seat of a mild degree of appendicitis.

There were, however, two disturbing factors. The height of the temperature and the degree of leukocytosis would have indicated a more virulent infection. Second, I had anticipated finding an appendix which lay in close enough proximity to the ureter to produce a secondary periureteritis. This certainly was not the case. The cecum was mobile and the appendix not only was not retrocecal, but being held to the upper part of the cecum by the adhesion could not even have assumed a deep-lying position. These two factors were sufficiently striking to make me realize that the appendix in this case was not the source of trouble. However, as you know, especially in those cases of appendicitis in which the offending organism is the streptococcus, the patient may have severe febrile and leukocytic reactions, and at operation the appendix itself may appear almost as benign as this one. Furthermore the slight swelling of the tip of the appendix with the definite kinking from the old adhesion made me think that perhaps a great deal of the pain might be explained on a mechanical basis.

A careful search up and down the right perivertebral groove revealed no abnormalities. The wound was then enlarged downward and medialward so that I could reach the bladder region. After doing this I was able to feel a mass which seemed to touch the extreme right tip of the bladder. This mass was easily brought into the wound and resembled a huge gangrenuos gallbladder. With gentle traction, however, the mass could easily be delivered and with it a loop of small intestine to which it was attached by a short, narrow, twisted pedicle.

It was evident that we were dealing with a Meckel's diverticulum which in some manner or other had become twisted upon its own base and had become straungulated. Thus the fever, leukocyte count, and exquisite right-sided abdominal pain and the pain which varied with bladder distention were clearly explained, as were also the quickness of the progress of the disease and the increase in intraperitoneal fluid.

The diverticulum arose from the antimesenteric border of the ileum, the base was about $\frac{3}{4}$ inch long, as thick as the average lead pencil, and was rotated once upon its own axis. The

gangrenous portion was about 3 inches long and $1\frac{1}{2}$ inches in diameter, tensely distended by fluid contents, and gangrenous as the result of a complete shutting off of its blood supply. The gut itself seemed normal except that the serosa was slightly injected.

The base of the diverticulum was ligated, a purse-string suture was placed around it, and after clamping the pedicle distal to the ligature, the diverticulum was amputated and the remaining stump inverted into the ileum. The amount of stump inverted was so small that there was no danger at all of stenosis

Fig. 136.—Photograph of diverticulum taken shortly after excision—two thirds normal size. The pedicle looks larger than it really was because it was crushed by the clamp in removal.

of the gut. The abdominal wound was closed in the usual manner, leaving a small Penrose tube drain in place.

The patient made an uneventful convalescence and was discharged from the hospital on the ninth day after operation.

Figure 136 shows the specimen shortly after removal. When the specimen was later split open the contents were found to be foul-smelling purulent fluid.

The exact explanation of the volvulus in this case is not possible, but what probably happened is that after a mild inflammation due to some unknown cause, the tip of the diverticulum became loosely adherent to the bladder by means of a plastic exudate. Once fixed at its tip, rotation could easily

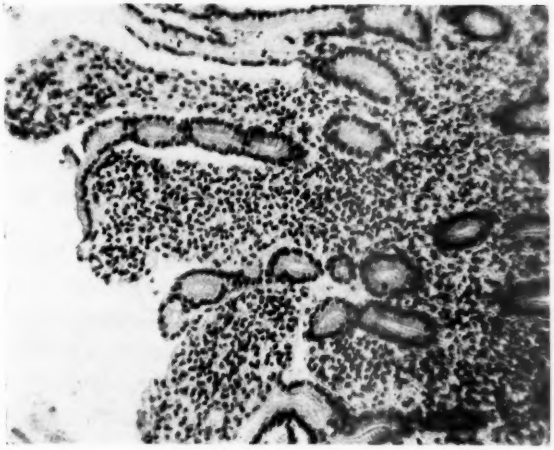

Fig. 137.—Photomicrograph of the tissue from the pedicle proximal to the site of torsion. The formation of the long tubules is suggestive of gastric mucosa, although the cells themselves are not.

Fig. 138.—Photomicrograph of the tissue taken from diverticulum distal to rotation.

follow a shift in position of the loop of ileum from which it sprang. According to Cullen, a volvulus in which twisting is

not rapid or tight enough to cause gangrene may produce a cystic dilatation with a flat pedicle. In this case after a certain amount of distention the twisting evidently became complete enough to shut off the blood supply and gangrene resulted.

FECAL IMPACTION DUE TO A MECKEL'S DIVERTICULUM

I wish to recall to your minds a case which Dr. W. M. Blum, who was then my house officer, and I reported a few years ago. This was a case of fecal impaction in a Meckel's diverticulum with a resultant intestinal obstruction.

The patient was a ten-year-old schoolboy who, except for a recent mild diarrhea, had been in perfect health up to four hours before admission to the hospital. While playing he had suddenly been seized with severe abdominal cramps. The pain at first had been generalized, but later became localized in the region of the umbilicus. He became nauseated and vomited several times. When he was first seen by me it was evident that the boy was extremely ill; his abdomen was slightly distended, there was a definite increase in muscle resistance, generalized abdominal tenderness, and a vague mass palpable to the right of the umbilicus. Although no exact diagnosis could be made, the most probable diagnosis seemed to be intestinal obstruction due to an intussusception.

At operation the large intestine, cecum, and distal part of the ileum were found to be collapsed. At a point about 8 inches from the ileocecal valve the intestine abruptly became distended, livid, and injected. At this point a broad-based pouch (evidently a Meckel's diverticulum) protruded from the antimesenteric border of the gut. The diverticulum and the intestine proximal to it for a distance of about 5 inches were distended by a doughy, semisolid mass. Above this the intestines were distended by gas and fluid. Gentle pressure was made on the diverticulum and the proximal intestine. Suddenly the semisolid contents slipped into the collapsed ileum followed by a gurgling of gas and fluid. The distended intestine almost immediately took on a more healthy appearance. We presumed that the diverticulum acting as a reservoir, had first been filled

with semisolid intestinal contents, and that then the pressure on the ileum had produced a functional stenosis which gradually led to a more and more firm impaction until a complete mechanical intestinal obstruction developed. Inasmuch as the obstruction had apparently been relieved and the proximal gut had resumed a nearly normal appearance, it was deemed wisest to interfere no further, but to close the abdomen, leaving the diverticulum in situ.

SYMPTOMLESS MECKEL'S DIVERTICULA

Twice I have seen well-defined Meckel's diverticula while operating for other intra-abdominal lesions, and once I have encountered a Meckel's diverticulum in a hernia sac. In every case the diverticulum was small, originated from antemesenteric border of the ileum with a broad base, and was apparently causing no disturbance. In each instance I left the diverticulum as it did not seem justifiable to complicate the operation by the removal of a congenital malformation which evidently neither in the past nor at present was affecting the patient.

DISCUSSION

It has long been known that a Meckel's diverticulum can be responsible for a variety of pathologic conditions.

Intestinal obstruction is frequently caused by a Meckel's diverticulum. Halstead years ago claimed 6 per cent of all obstruction cases were due to this deformity. Horine found 4 cases in a series of 100 consecutive cases of acute intestinal obstruction were due to Meckel's diverticulum. Some other authors quote about the same figures. The obstruction may be brought about in various ways. The diverticulum is frequently attached to the umbilicus, the abdominal wall, or intra-abdominal viscera at its tip or by a fibrous filament, and loops of bowel may slip over or under this band and become incarcerated and strangulated. The diverticulum may actually become wrapped around a loop of bowel and thus cause an obstruction. The diverticulum may produce a torsion or volvulus, the loop of ileum from which it springs. Obstruction may be pro-

duced by an intussusception. Paralytic ileus may result from a peritonitis secondary to a diverticulitis.

Inflammation of a Meckel's diverticulum is not unheard of. The inflammation may proceed to gangrene and perforation.

Intussusception may start with a Meckel's diverticulum. In cases in which the diverticulum opens through the umbilicus the intussusception may become external.

Intestinal hemorrhage, especially in children, may originate in an erosion in a Meckel's diverticulum. Abt and Strauss reported 3 cases. Others have been reported.

Ulceration of the diverticulum may occur. Occasionally gastric mucosa may be found in a diverticulum and ulcers resembling peptic ulcers have been described.

A fecal impaction in the small intestine was caused in the case I just described indirectly by a Meckel's diverticulum.

Volvulus of a Meckel's diverticulum, such as I have here reported, is not common, but, on the other hand, is not unknown. Many have been reported. I made an attempt to compile cases of volvulus of Meckel's diverticulum reported in the literature, but abandoned it as it became more and more evident that in far-advanced cases it would be impossible to distinguish on the basis of case reports those in which the gangrene was due to an acute infectious process from those in which the primary condition was mechanical.

Tumors of Meckel's diverticulum have been reported. These have been of an infinite variety, inasmuch as almost any type of tissue rests may be found included in Meckel's diverticulum.

A Meckel's diverticulum usually arises from the antimesenteric border of the ileum about 12 inches from the ileocecal valve. It usually has a base as broad as the lumen of the gut from which it arises. Its wall usually contains all the layers of the intestine. Diverticula have been described in which the muscularis is absent and these have been termed "acquired diverticula," and may be a herniation of the lumen of the gut through a weakness in the wall. These diverticula frequently arise from the mesenteric border of the intestine and may dissect their way into the mesentery and endanger

the blood supply of neighboring portions of the bowel. Meckel's diverticulum is most frequently found in males.

It is not surprising that a Meckel's diverticulum should give rise to the same pathologic conditions as the appendix or any other diverticulum of the intestine. Because of the possibility of these pathologic complications there has been a growing tendency to advise removal of any diverticulum found by chance at operation, in the same manner of reasoning which prompts many surgeons to remove the appendix whenever possible at any laporotomy.

It is an extremely significant fact that anatomists and pathologists, such as Cunningham, Adami, Kauffmann, etc., report the incidence of Meckel's diverticulum to be a little over 2 per cent, while in various surgical clinics it is so seldom encountered: McGlannan, 3 cases in 1400 laparotomies; Balfour, 15 cases in 10,000 laparotomies, etc. This shows that although a Meckel's diverticulum is a fairly common congenital abnormality, yet as a causative factor of disease it is not of considerable moment.

For this reason I advise against a routine removal of a Meckel's diverticulum encountered as a chance finding during an abdominal operation for some other pathologic condition.

In children there is perhaps more justification for a prophylactic removal than in adults. The experiment as to whether or not the diverticulum is going to cause trouble has not been answered by the evidence of years.

There are two types of Meckel's diverticulum which are most apt to cause harm. The first is the diverticulum which is attached to the abdominal wall or some intra-abdominal viscus either by its tip or by a fibrous band, the second is the diverticulum which does not arise from a broad base, but from a narrow pedicle. The possibility of danger from diverticula of these types is sufficiently high that their removal for prophylactic rasons might be justified provided, of course, the pathologic condition present or the nature of the laparotomy do not contraindicate this additional procedure.

A broad-based diverticulum is best removed by an elliptical

incision parallel with the bowel and sutured transversely. In the case of a narrow pedicle the procedure is similar to that used for removing the appendix.

No general rules can be laid down for the management of cases in which the Meckel's diverticulum is the cause of illness. In inflammatory conditions removal is obviously indicated, as in the first case I reported. In certain instances in which the diverticulum causes mechanical interference it may be best to leave it alone, as in the second case here recorded. Furthermore, the technic of removal will vary. In the first case the diverticulum was removed in the same manner as an appendix. In the second case I had considered drawing the diverticulum through the abdominal wall, thus exteriorizing it, then amputating the diverticulum, leaving the stump open and draining the lumen of the ileum. This procedure was unnecessary when it was found that the wall of the diverticulum was normal.

SUMMARY

A case has been presented in which a Meckel's diverticulum having become rotated on its own pedicle, became strangulated and led to a chain of symptoms which simulated those produced by inflammation of the appendix. The condition was discovered at operation and the diverticulum removed.

A second case was recalled in which an intestinal obstruction resulted from a fecal impaction starting in a Meckel's diverticulum. In this case the diverticulum was not removed.

Three other cases were cited in which a Meckel's diverticulum was an accidental finding during operation and in which the diverticulum had never caused any disturbance. In all 3 cases the diverticula was left in situ.

A list of complications which may be caused by Meckel's diverticula has been presented. This list is long and dire.

In view of the fact that the vast majority of individuals who harbor a Meckel's diverticulum are never disturbed by this interesting and not so uncommon congenital abnormality, I feel that it is unwise to recommend the routine prophylactic removal of this deformity when found by chance during laparotomy.

Meckel's diverticula which are attached at their tip either directly or by a fibrous band, as well as those diverticula that have a narrow pedicle, are the most dangerous types. The prophylactic removal of such types is justified, provided this additional procedure does not dangerously complicate the laparotomy. In the first of these types the danger of intestinal obstruction from incarceration or torsion is particularly great. In the second type the danger of inflammation is present and the removal of the diverticulum is particularly simple.

CLINIC OF DR. EDWIN M. MILLER

PRESBYTERIAN HOSPITAL

TWO CASES OF STRANGULATED HERNIA DUE TO RUPTURED APPENDIX. OPERATIVE REPAIR OF SUPRACONDYLAR FRACTURE OF THE HUMERUS IN A CHILD

I SHOULD like to present for your consideration 2 rather unusual and, to me, extremely interesting cases in elderly gentlemen of about the same age, almost identical as far as clinical aspects are concerned, and presenting features of interest from the standpoint of diagnosis, pathology, and treatment.

Ordinarily when one is called upon to see a patient complaining of a painful irreducible swelling in the inguinal or femoral regions associated with nausea and vomiting, no matter if it is in a child or adult, one has a right to assume that a strangulated hernia is present, but after the experience with these 2 cases I am convinced that one might easily be mistaken. Both of these men present such a clinical picture.

One, aged sixty-nine, was first seen on April 13, 1929 by Dr. James Murray Washburn at the patient's home, where he was attempting to carry on his duties as a butler. He had not been feeling very well for about five days and complained of nausea and pain in the region of the right inguinal hernia, which he had had for many years. A diagnosis of strangulated inguinal hernia was made and he was sent to the hospital. On entrance his temperature was 102 F., leukocytes 18,600, a tender irreducible mass was found in the right inguinal region, slight abdominal distention, and muscle rigidity, particularly over the right side. He was immediately sent to the operating room. Through the usual oblique incision (Fig. 139), directly over the protruding mass, the inguinal canal was opened, exposing markedly edematous tissue in the neighborhood of a direct hernia

the size of a large goose egg. Upon opening the sac, much to my surprise, it contained no loop of bowel, nothing but omentum, which was greatly swollen and covered by a plastic exudate. It was evident at once that we were dealing with no ordinary case of strangulated hernia of the bowel, but with pathology within the hernial sac, which must be secondary to the trouble within the general abdominal cavity. After bluntly reducing this

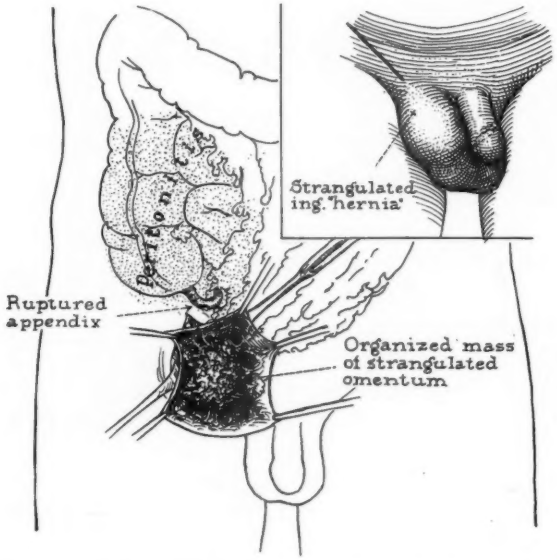

Fig. 139.—Incarcerated hernia of the great omentum secondary to a ruptured appendix with peritonitis. Age sixty-nine years. Recovery.

swollen omentum and leaving the inguinal wound entirely open temporarily, the abdominal cavity was entered through a lower right rectus incision. A plastic inflammatory exudate was seen everywhere. The inflamed omentum was reflected medially, exposing an area of peritonitis involving the entire quadrant. Upon separating the adherent loops of inflamed bowel, a perforated appendix was found lying medial to the cecum and extending downward into the pelvis. Appendectomy was done,

the ligated stump buried beneath a catgut purse string, and adequate drainage established both through the rectus incision itself, and through the open hernial sac. No repair of the inguinal canal was attempted. The recovery of this man, who, as you see, is now in good health and has long since returned to active duty, in view of the fact that a pneumonia marred his postoperative course, has been extremely gratifying.

The features of the second case are not materially different from the first. This gentleman, aged seventy-four, was first seen at his home by Dr. Frank Kelley on June 10, 1929. He had been sick for about three days with rather diffuse abdominal pain, some nausea, and a little vomiting. An irreducible mass was present in the right femoral region, about the size of a goose egg. A diagnosis of strangulated femoral hernia was made and he was at once sent to the hospital. Upon admission his temperature was normal, leukocytes 13,800. He was sent to the operating room immediately. Through an oblique incision (Fig. 140) directly over the mass in the region of the femoral opening a hernial sac the size of a goose egg was found. The fat was dissected away from the sac and the fundus broken into bluntly, allowing perhaps 2 ounces of thick yellowish-gray pus to escape. The lining of the sac was necrotic and no loop of bowel was present. It was quite evident here also that we were dealing with no ordinary strangulated hernia, but rather with a suppurative process which had originated within the abdomen and had passed through the femoral canal into the hernial sac. The abdomen, therefore, was opened through a separate right rectus incision, revealing an extensive peritonitis involving all of the loops of the bowel in the right lower quadrant. These loops were separated one from the other until an abscess was located in the depth of the pelvis containing fully $\frac{1}{2}$ pint of thick yellow pus, which was aspirated away as fast as it was encountered. The cecum was followed down and a perforated appendix was found with an opening it it 3 mm. in diameter near the base. The appendix was removed, but the stump could not be invaginated. When the pus in the field was thoroughly aspirated away, an ileostomy was made in the loop of small

bowel nearest to the mass involved in the inflammatory process in order to prevent, if possible, or at least help relieve, a further paralytic ileus. Adequate drainage was established here as in the other case, not only through the rectus incision itself, but through the open femoral hernial sac. In this case also the re-

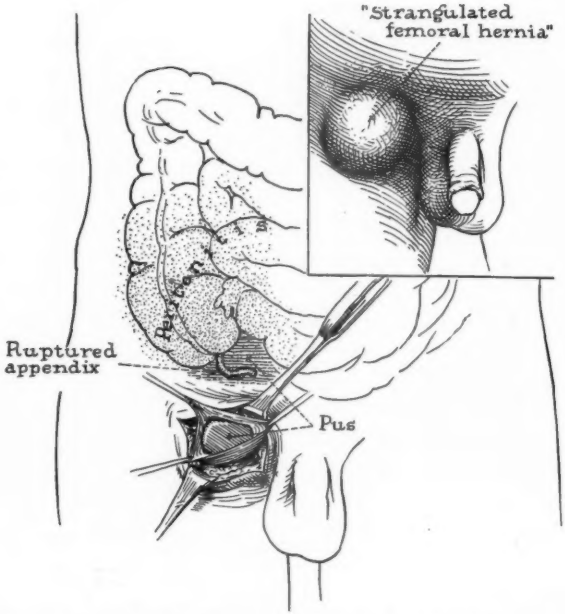

Fig. 140.—Abscess within the sac of a right femoral hernia secondary to a perforated appendix with extensive peritonitis.

covery in view of the extensive pathologic findings seems rather remarkable.

The third case which I wish to present to you is that of a little girl four years old. She was brought to the hospital August 28, 1929 with a supracondylar fracture of the left elbow of three weeks' standing. There was a marked deformity present. The tissues about the elbow were discolored from the infiltration of blood and the lower edge of the long upper bone

fragment could be felt anteriorally just under the skin, ½ inch below the crease of the elbow. Flexion was limited to 90 degrees. The *x*-ray (Fig. 141) revealed marked anterior displacement of the upper fragment and newbone formation bridging the gap to connect with the short lower fragment in its normal position. There was no evidence of nerve injury nor signs of beginning ischemic palsy. As far as treatment was concerned two possibilities were considered: first, to approach through a posterior

Fig. 141.—Supracondylar fracture of three weeks' duration in a girl aged four years with displacement of the upper bone fragment anteriorly almost perforating the skin. New bone formation along the line of the old shaft. Soft tissues about elbow extremely indurated. Flexion limited to 90 degrees.

incision, chisel away the rapidly developing new bone and reduce the displaced upper fragment of the humerus into its normal position; or second, to leave the newly formed bone intact to serve as the permanent new shaft and to remove by open operation a portion of the old shaft which lay anterior to the normal line of the humerus. After due consideration of all the factors present, the latter plan was decided upon. The question then remained whether to operate at once, going through tissues that were very much indurated and at a time when the newly develop-

ing shaft was still quite thin, or whether to wait for a period of a month or more until the soft parts had regained their normal consistency and the new shaft had attained the thickness and strength of a normal bone. The latter course was followed. After five weeks had elapsed another *x*-ray picture was made (Fig. 142) revealing a well-developed new shaft directly in line

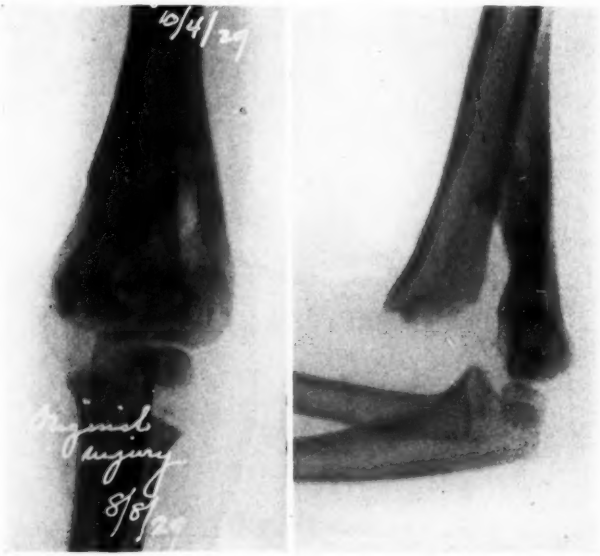

Fig. 142.—Picture made about five weeks later, showing the increase in thickness of new bone, sufficient to serve as a new permanent shaft. Old displaced shaft to be removed through an anterior incision along the dotted line.

with the normal humerus. The soft parts about the elbow had lost their induration and we proceeded with the operation. Through an anterior incision directly over the displaced lower end of the humeral shaft the excess bone was removed with a Gigli saw, as illustrated in the accompanying picture. The result thus far has been very satisfactory. While the anterior surface of the humerus was not perfectly smooth through the

line of section of the bone (Fig. 143), it in no way interferes with the flexion of the elbow, which is now complete.

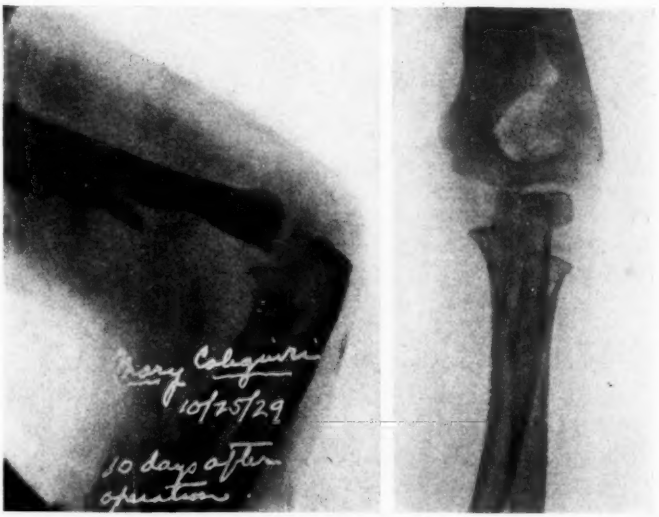

Fig. 143.—Appearance of the bone ten days after operation. Flexion of the elbow now complete. Anterior surface of the humerus not as smooth as one would like to see it, but in no way interfering with function.

It seems to me that this case illustrates what a good result may be obtained in a bad fracture if a little conservatism is used.

CLINIC OF DR. C. B. HUGGINS

ALBERT MERRITT BILLINGS HOSPITAL

HYDRONEPHROSIS (FOUR CASES)[1]

THIS morning I wish to present 4 patients who have been operated on in the last six months for hydronephrosis. These cases illustrate well many of the practical points concerned in the diagnosis and treatment of this disease as well as variations in pathology encountered at operation.

The symptomatology of hydronephrosis may roughly be divided into four groups:

1. That class of patients with classical renal symptoms.
2. That class without renal symptoms, but with symptoms caused by pressure of the kidney on adjacent viscera.
3. A class in which symptoms are purely generalized, due to either renal insufficiency, in which there is usually a bilateral process; or infection in which the constitutional symptoms make slight or absent local renal symptoms.
4. A group of patients without any symptoms.

EARLY AND ADVANCED HYDRONEPHROSIS DUE TO ACCESSORY RENAL VESSELS CAUSING LOCAL RENAL SYMPTOMS

Case I (9973).—This young man, aged nineteen, visited the clinic complaining of attacks of renal pain. Two years ago he fell a distance of 7 feet, with immediate pain in the right kidney. There was spasm over the right kidney and hematuria at this time. The referring physician told the patient that he had a dislodged kidney and treated him by adhesive strapping of the loin, and bed rest, with subsidence of the pain in about one week.

Symptoms of severe pain in the right loin and under costal

[1] This is part of a clinic presented before the American College of Surgeons, Chicago, October, 1929.

margin recurred in six months; the pain was without radiation, and was of enough intensity to require morphine, which relieved the pain for several hours. Later heat gave some relief. In the past eighteen months he has had three similar attacks, each requiring bed rest from one to seven days. There has been no

Fig. 144.—Case I. Pyelogram of the right kidney. Note the dilatation of the pelvis and calyces and the sharp angulation at the ureteropelvic junction at the crossing of the vessel.

radiation in any attack. In the last six months he has had a frequent dull aching pain in the loin, which is just noticeable.

Physical examination was negative on admission. There was no tenderness or pain on palpation of either renal area. Neither kidney was palpable. The urine was negative. Phenol-

sulphonephthalein excretion was normal. Roentgen studies of the renal areas were negative.

Cystoscopy was done and disclosed a normal bladder; both ureters were catheterized; the left ureter easily; the right meeting an obstruction 24 cm. from the ostium. The divided urines were essentially negative. Phenolsulphonephthalein was excreted from the left side in excellent concentration in four

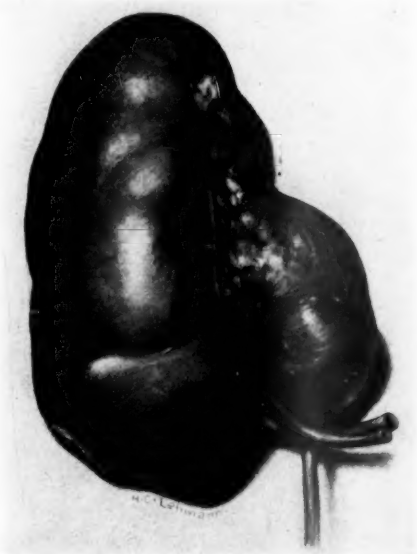

Fig. 145.—Case I. Drawing of the excised kidney and ureter, demonstrating the anatomical situation found at operation.

minutes; from the right kidney there was no excretion in twelve and one half minutes. Pyelograms were made disclosing a normal left kidney. The pyelogram of the right kidney (Fig. 144) showed a gross dilatation of the kidney pelvis and of the primary and secondary calices. There is an acute angulation of the ureter at its junction with the kidney pelvis (Dr. C. S. Capp). Diagnosis was made of hydronephrosis due to an accessory renal vessel. Exploration of the kidney was done one week later

and revealed a 50 per cent increase in size of the right kidney. There was a marked swelling of the extrarenal portion of the pelvis and at the ureteropelvic junction anterior to the ureter there was found a large artery about the size of the radial artery which pulsated noticeably. Even after lysis of the kidney it was impossible to milk the urine from the distended pelvis, using as much pressure as will empty a normal gallbladder. Nephrec-

Fig. 146.—Case I. Injection of the aberrant artery with sodium iodide, showing the amount of kidney tissue supplied by the artery.

tomy was done. Convalescence was smooth. Since then the patient has been relieved of pain.

Examination of the specimen (Fig. 145) showed a normal ureter; dilated kidney pelvis; possibly 50 per cent reduction of renal parenchyma. There was no evidence of valve formation or stricture at the ureteropelvic junction. The accessory lower polar artery was injected with a solution of sodium iodide, soon

after operation (Fig. 146). It demonstrated that the vascular supply of the abnormal vessel comprised roughly the lower third of the kidney.

Comment.—This is a text-book case of an aberrant lower pole vessel causing hydronephrosis. The most interesting feature is the large size of the accessory vessel and the large amount of kidney tissue supplied by it. Since it has been abundantly established (Hinman-Heplar and others) that the renal arteries are end-arteries and these do not anastomose to form compensatory circulation, it follows that ligation would have produced anemic necrosis of the lower third of the kidney. If the kidney tissue were known to be sterile, bland infarction would probably result; but if it were infected, and there is considerable evidence from the work of David, Adami, the writer, and others that the normal kidney of dogs is frequently infected, more serious results would ensue. Since ligation is out of the question then in this case, it seemed that kidney suspension or pyeloplasty were too radical forms of treatment to be followed, so that nephrectomy was done.

Case II (13604).—This man is twenty-four years of age and for ten years has had pain in the right side of the abdomen. In 1919 he had pain in the right lower quadrant of the abdomen extending to the loin, lasting for two days. Since then he has had an average of ten attacks a year, with sometimes as much as a year's interval between attacks, and sometimes as little as a week. Recent attacks have lasted seven to ten days and have been associated with headache and nausea. Pain was relieved to some extent during the attacks by the knee-chest position. A "gnawing pain" is felt in the right loin at all times. Appendectomy at age eleven. No bladder symptoms or gross changes in urine.

Physical examination showed tenderness in the right costal margin with a renal mass extending to appendectomy incision anteriorly. Cystoscopy was negative except for blockage of the ureter at the right ureteropelvic junction. A fair flow of clear colorless urine from the right catheter; no excretion of dye in

sixteen minutes. Yellow concentrated urine from left side with normal dye excretion. Pyelograms were made; the severe pain was reproduced by injection of 4 cc. of sodium iodide. This pain persisted for three days; the second and third day after cystoscopy were characterized by a temperature of 100 F. and leukocytosis.

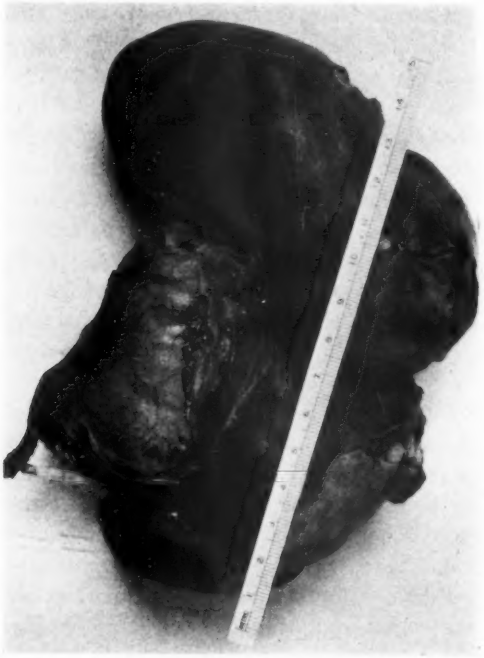

Fig. 147.—Case II. Large hydronephrosis. The thickness of the cortex is seen: the ureter is normal.

At operation a moderately large kidney was encountered. A trocar was inserted, and about 400 cc. of cloudy urine containing pus was evacuated. This was followed by collapse of the kidney; it was then easily dissected out by gauze dissection from surrounding tissues. An aberrant blood vessel was seen entering the kidney 2 cm. below the main vascular pedicle and it was

ligated. Closure in layers with drainage. Uncomplicated convalescence.

Examination of the operative specimen demonstrated that the pelvis and calyces were markedly dilated, reducing the functioning renal tissue to about 4 mm. in thickness. The ureter was normal; there is a sharp change between the normal ureter and the dilated pelvis at the exact crossing of the pelvis by the vessel which was anterior to it (Fig. 147).

Comment.—This is a more advanced stage of hydronephrosis due to aberrant vascular supply of the kidney. The kidney became infected after cystoscopy and pyelography. This has been a not unusual occurrence in these large hydronephroses and it is our feeling that in these cases instrumental study of the urinary tract should not be undertaken unless the situation can be adequately dealt with if this complication should arise.

Reproduction of the exact pain is an important diagnostic sign. It has been our experience that in many of these cases the catheter cannot be introduced into the pelvis, so that aspiration of large quantities of fluid is not feasible. The principal diagnostic aid in any case will be the pyelogram; even if a large quantity of fluid is obtained by catheter, it is distinctly unwise to replace the fluid by a similar quantity of the pyelographic medium, since it may make trouble in the stagnant kidney. A small amount of sodium iodide will by diffusion give excellent diagnostic plates, and is safer.

Case III (13349).—This man of seventy-six entered the clinic in July, 1929 complaining of a large mass in the left upper quadrant of nine years' duration accidentally discovered in 1920 during an osteopathic manipulation. It gradually grew from about the size of a lemon to the size of a football without any pain locally. During the last two years he has had a feeling of weight in the left side of the abdomen, and has suffered from dyspnea at times.

Examination showed that the mass filled most of the left side of the abdomen; its circumference anteriorly measured 30 cm. roughly. It transmitted bimanual pressure from loin to

anterior abdominal wall. The general examination showed that this man was in excellent general shape. The urine was negative.

A diagnosis of pancreatic cyst was temporarily made. Cystoscopic examination showed the ureters in normal location; the entire left side of the trigone was atrophied markedly as compared with the right. The left ureter was small and would not admit the insertion of a No. 4 F. ureteral catheter. Indigo

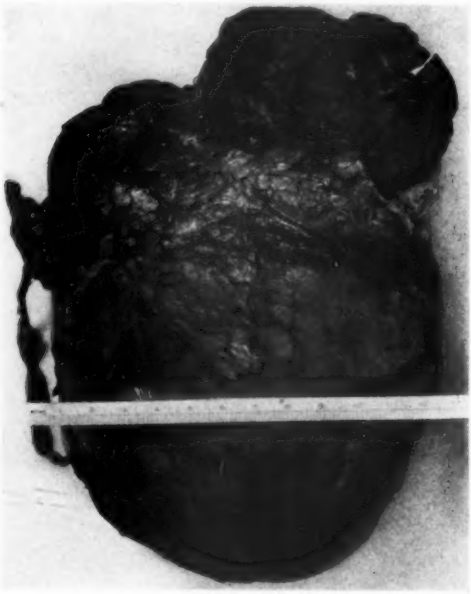

Fig. 148.—Case III. Large hydronephrosis. The ureter is normal.

carmine intravenously was excreted normally from the right ureter, but did not appear in twenty minutes from the left side. A right pyelogram was normal.

Diagnosis of left-sided hydronephrosis was then made on (1) fluctuant character of the tumor, (2) easy bimanual palpation, (3) atrophy of left half of trigone with absence of evidence of excretion of dye from the left kidney.

Operation through a loin incision demonstrated a very large hydronephrotic kidney extending from the diaphragm to the true pelvis, and medially on top of the great vessels to the right of the midline. Puncture of the cyst lead to easy removal of it by gauze dissection. An aberrant artery was present crossing behind the ureter leading to the lower pole; this led to a little difficulty in control of the hemorrhage. Suture of the wound in four layers with drainage. Uneventful convalescence and discharge from the hospital on the twenty-third postoperative day.

Examination of the specimen (Fig. 148) revealed no obstruction to the insertions of a fine probe in the lumen of the ureter (14 cm. excised). Both the lumen and musculature of the ureter were decreased in size; a sharp contrast occurred between the atrophy of the ureter and the markedly dilated kidney. There was no evidence of valve or stricture formation at this junction, so one is forced to the conclusion that the obstruction was from without, and since an abnormal vascular supply was encountered to the lower pole, that this was the cause of the hydronephrosis.

Comment.—This was an unusual case as far as age is concerned; hydronephroses first coming to notice at the age of sixty-seven, apart from those cases due to stone, prostatism, or tumor of the bladder are distinctly rare. Similarly the aberrant vessels will usually cause trouble before this age. Also it brings to mind the very significant importance of the finding of hemiatrophy of the trigone, indicating as it usually does a kidney whose excretory function has been blocked for years.

SUMMARY OF THE ABERRANT RENAL VESSEL QUESTION

We can summarize here in more or less tabular form some of the important points concerning the truant vessels to the kidney.

Anomalous renal vessels are of surgical importance for two reasons: (1) Their association with hydronephrosis as the direct etiologic agents; (2) a source of unexpected hemorrhage during renal operations.

Frequency.—They occur in a large percentage of patients without symptoms or any presenting evidence of their presence. (1) Quain's Anatomy, present in 20 per cent of all cadavers;

(2) Eisendrath and Strauss, 21 per cent; (3) Eisendrath, main renal vessel origin can be expected 1 : 5; (4) Seldowitch, 43 cases in 150 specimens or 30 per cent of these 10 cases were bilateral; 33 cases unilateral; (5) Squier, it is almost the rule that kidneys with fetal lobulation have abnormal vessels; (6) Mayo-MacCarty-Broders, in 18 out of 20 cases the vessel arose from main renal; (7) Ekehorn, vessels posterior to ureter in 28 per cent of cases of aberrant vessel; anterior in 64 per cent arteries are more common than veins.

Etiology.—The current conception depends on the embryological concept of Jeidell and of Bremer that the kidney arising in the bony pelvis ascends and rotates to its characteristic loin location, and during its course reception and obliteration of blood vessels from the aorta take place seriatim. The vessel can be thus explained by failure of obliteration.

Obviously vessels supplying the upper pole of the kidney cannot obstruct. The obstructive phase is probably due to one of three obvious factors in explaining the hydronephrosis due to lower pole vessels: (1) Movable kidney descending over the renal vessel; (2) adhesions between ureter and vessels; (3) tension due to insertion of the vessel.

Albarran dissents in the view that aberrant vessels are the cause of hydronephrosis. "Whether the abnormal vessels pass in front of or behind the ureter, one is unable to understand how it is able to cause hydronephrosis. On the contrary, a hydronephrotic kidney may descend and the ureter become encroached upon by vessels which cross it from in front or behind."

Symptoms.—The symptoms of any disease may be none (Richard Cabot).

1. Attacks: Dull to sharp pain at intervals in loin; feeling of fulness in the loin between attacks. In our experience the pain has not radiated into groin or testicle.

2. Rest in bed and genupectoral position often eases the pain.

3. Age: Usually symptoms occur before age twenty-two (Braasch).

Diagnosis.—1. In early stages the kidney is frequently not palpable, although it may be in a standing position of the

patient. In later stages a cystic kidney is usually palpable bimanually.

2. Distention of the pelvis usually reproduces the pain (Kelly).

3. Pyelogram is characteristic of this disease.

Treatment.—1. Nephropexy (Kelly). However, Rafin and Quinby describe cases where it failed.

2. Ligation of vessel. In 13 ligations carried out during the treatment of 20 cases by Mayo *et al*, the pain was completely relieved.

3. Ureteral section and reimplantation (Quinby).

4. Nephrectomy.

It is the author's opinion that if the vessel is small, ligation plus nephropexy is indicated. If a large vessel and the opposite kidney is normal by urological study, nephrectomy should be carried out. If there is a bilateral process and large vessels the operation advocated by Quinby is indicated. A large hydronephrotic kidney associated with a small vessel should also be treated by nephrectomy.

ADVANCED HYDRONEPHROSIS WITH SYMPTOMS ENTIRELY CAUSED BY PRESSURE ON ADJACENT VISCERA

Case IV (16427).—This last patient, a painter, is thirty-five years of age; he consulted the clinic because of a long story of indigestion and epigastric distress dating back seven years. He has spent two and a half years of this time on various types of ulcer management without relief and has had a great many studies done by various doctors.

Seven years ago he gradually began to have hunger distress in the abdomen several hours after eating, relieved by alkalies and food taking for a while. The distress was localized to the epigastrium. *x*-Ray studies at this time were negative. A Sippy diet gave complete relief for six months; after several months of distress he consulted physicians, had gastric aspirations made, *x*-rays, etc., and was again placed on a diet. This regular cycle of events occurred almost annually. There is at times marked constipation.

In the last month there had been a good deal of vomiting and the distress had been unrelieved by eating or alkali. Physical examination indicated a thin sparse man with slight jaundice present in sclera and skin. Inspection of the abdomen reveals a definite bulging on the right side both in the right lower quadrant and right upper quadrant. A cystic tumor is felt in

Fig. 149.—Case IV. The stomach, duodenum, and colon, injected with barium, are seen markedly displaced to the midline by a large hydronephrosis of the right kidney.

Fig. 150.—Case IV. Pyelogram of the right kidney. Compare with Fig. 149.

this region and pressure is transmitted bimanually between loin and anterior abdominal wall.

Laboratory examination: The van den Bergh test gave a marked delayed reaction. Ewald test gave 60 units of free acid and 82 of total acid in the stomach contents. Nonprotein nitrogen of the blood was 54.5 mg. per 100 cc. The urine was negative. Phenolsulphonephthalein output 40 per cent in two hours.

x-Ray studies: (1) Nonvisualization of the gallbladder

(2) the stomach and duodenum lay entirely to the left of the midline (Fig. 149); (3) barium enema showed that the entire colon lay to the left of the midline.

Cystoscopy was done and the bladder was negative. The left ureter was easily catheterized. The right ureter could be

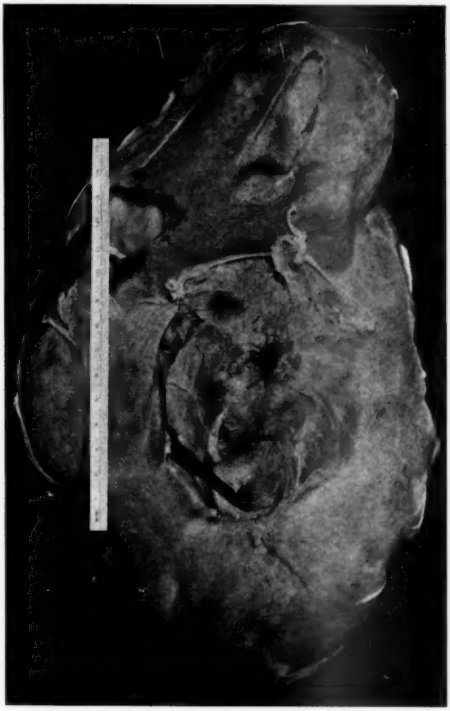

Fig. 151.—Case IV. Tremendous hydronephrotic kidney. The ureter (see arrow) is normal. No demonstrable cause of the hydronephrosis.

catheterized 10 cm. with a No. 6 F. olive-tipped catheter. A No. 7 Blasucci catheter passed 25 cm., but very slowly for the entire distance; it did not drain. The urine and function of the left kidney were normal. Pyelograms were made; the left kidney was normal. The right kidney was injected with 15 cc.

of a 10 per cent solution of sodium iodide and the patient suffered a feeling of fulness in the loin, but no pain. The right pyelogram (Fig. 150) demonstrated a large hydronephrosis. About twenty-four hours after pyelography the patient had considerable pain in the right kidney requiring morphine; this

Fig. 152.—Case IV. Cholecystogram seventeen days after removal of large hydronephrosis. Previous cholecystogram showed nonfunction of the gallbladder.

was present for two days and was associated with fever and leukocytosis.

Operation through a loin incision revealed a very large kidney (Fig. 151) extending from the diaphragm to the true pelvis and to the left of the midline. Nephrectomy was done after puncture of the tumor. Convalescence was uneventful. The patient has had no further distress, vomiting, or constipation.

x-Rays were repeated on the seventeenth postoperative day and showed (1) a normally functioning gallbladder by the Graham test (Fig. 152), (2) stomach and duodenum in normal location, (3) colon in its normal location (Fig. 153).

Comment.—This is a case of a very large hydronephrotic kidney producing symptoms entirely from pressure on adjacent viscera. The jaundice is a rather uncommon feature; since there

Fig. 153.—Case IV. Barium enema eighteen days after right nephrectomy for tremendous hydronephrosis. Note return of colon to normal anatomical location. Compare with Figs. 149, 150.

was nonvisualization of the gallbladder it can probably be safely explained by pressure on the ducts.

It is very interesting also that the viscera should return so quickly to an absolutely normal anatomical position after operation.

The etiology of the hydronephrosis in this case is unknown. There is no abnormality of the vascular supply, valve formation, stricture, stone, or tumor on close examination of the specimen.

The ureter is normal; it is patent to the examining probe and dilatation begins at the ureteropelvic function. Undoubtedly more exact information that can be derived only from the autopsy table is needed before we can unravel many of these hydronephroses.

The choice of incision need give us little concern in the management of these large hydronephroses; the usual oblique lumbar loin incision gives good access to the kidney. The important factor in their operative management is early puncture of the kidney, letting out the contents. This so facilitates operation that usually the kidney can be removed by gauze dissection. The vascular supply to these kidneys is smaller than normal and gives no trouble. Careful observation of the patient's blood pressure in 7 of these patients with large hydronephrosis has demonstrated no fall in blood pressure concomitant with sudden evaucation of the urine, such as has been described with sudden emptying of a chronically overdistended bladder.

BIBLIOGRAPHY

Bremer, J. L.: Amer. Jour. Anat., 18, 179, 1915.
Eisendrath: Ann. of Surg., 71, 727, 1920.
Eisendrath and Strauss: Trans. Amer. Urol. Assoc., 4, 178, 1910.
Jiedell: Anat. Record, 5, 47, 1911.
Mathè: Jour. Urol., 19, 211, 1928.
Mayo, Braasch, MacCarthy: Jour. Amer. Med. Assoc., 52, 1383, 1909.
Quinby: Jour. Urol., 10, 45, 1923.
Rafin: Trans. Assoc. Franç. d'Urol., 153, 1906.

CLINIC OF DR. CHESTER C. GUY

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PAGET'S DISEASE: ITS DIFFERENTIATION FROM METASTATIC CARCINOMATOSIS OF BONE

I WISH to present 2 unusual cases which are perhaps of special interest because of the similar urological and roentgenological findings in each. Both of these patients came to the Illinois Central Hospital, one to the surgical service of Dr. W. T. Harsha and the other to the medical service of Dr. L. H. Sloan, and I wish to express my indebtedness to these men for the privilege of studying and reporting them. Both cases were at first incorrectly diagnosed, because we were led astray by the *x*-ray appearance of the pelvic bones. We interpreted the bone changes as indicating an extensive metastatic carcinomatosis and this diagnosis seemed particularly tenable because both men were of advanced age and both had prostate glands which were enlarged and felt suspiciously like malignant tumors.

I will briefly review their case histories. Mr. S., age sixty-two years, entered the hospital in March, 1927 because of an aching pain in the right lumbar region of a few days' duration, Routine physical examination revealed nothing of importance except an enlarged, stony hard, nodular prostate, which felt like carcinoma to the examining finger. *x*-Ray pictures of the pelvic bones showed a mottling which was very suggestive of carcinoma metastases. The urine was entirely normal and there was no history of any urinary tract disturbances. A diagnosis was made of primary carcinoma of the prostate with metastases to the pelvic bones, and he was given two deep *x*-ray therapy treatments.

After a month in the hospital he was relieved of his pain and returned to work. Over a year later, in August, 1928, he

reappeared at the hospital because of a recurrence of the pain in the right lumbar region. He also then complained of some headache and occasional dizzy spells. *x*-Rays of the skull were made, and the typical fuzzy outlines, so characteristic of Paget's disease, were seen. Urinalysis was again negative and re-examination of the prostate revealed it to be small, soft, and entirely normal on palpation. The change in the prostate is probably best explained on the basis of a chronic inflammatory process which had subsided. Certainly, at the last examination, there could be no suspicion that it harbored a malignant tumor.

The second patient, Mr. C., aged seventy-one, entered the hospital in May, 1928, complaining of urinary retention of five days' duration, which necessitated catheterization for relief. He had also suffered from vague pains in the back and legs which he said caused him to assume a slight stooping position. Fourteen hundred cc. of urine were removed by catheterization, after which, on rectal examination, the prostate gland was felt to be enlarged and rather boggy. Urinalysis showed only a moderate amount of pus. Blood urea and creatinine determinations were normal. An *x*-ray of the pelvic bones revealed a generalized mottling which was supposedly due to tumor metastases from a malignancy of the prostate. Through a suprapubic incision the prostate was exposed and we found an enlargement of the median lobe to be the cause of the urinary retention. But the gland looked and felt benign, and a microscopical study of the median lobe, which was removed, failed to reveal any carcinoma. Search for some hidden malignancy elsewhere was unsuccessful. The prostatectomy wound healed well, and he was discharged home with a guarded prognosis. He returned in six months with no urinary complaints, but at this time the bowing of the back and the head deformity were noted, and *x*-rays of the skull made on the suspicion of Paget's disease.

Now, when we examine these patients, we find that both have a definite bowing of the legs and a flattening of the back of the skull, which we can detect by palpation. Also notice that Mr. C. is stoop-shouldered from an anterior bowing or

lordosis of the spine. These deformities are not as marked as in many cases of Paget's disease, but they are sufficiently marked so that they are very suspicious of the disease if not actually

A

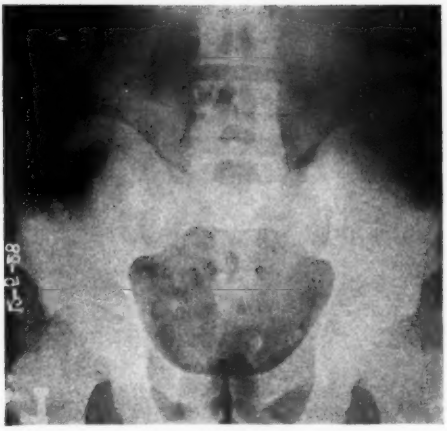
B

C

Fig. 154.—A, Normal skull. B and C, Skulls of two patients with Paget's disease.

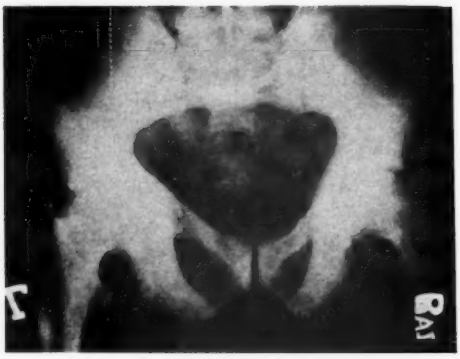
diagnostic, especially when considered together with the bone changes as shown by *x*-ray examination.

The *x*-rays are typical of Paget's disease and illustrate the peculiar changes of the bones in this condition. You will note in the pictures of the pelves of both patients, the alternate



A

B



C

Fig. 155.—A, Normal pelvis. B and C, Pelves of two patients with Paget's disease.

areas of increased and decreased density, the bone above the acetabula being much denser than normal. The changes also appear in the lower lumbar vertebrae, but are less well marked. Carcinoma causes bone rarefaction and destruction with little or no increase in bone density, but the differential diagnosis from Paget's disease in the pelvis may be very difficult or impossible.

In the skull pictures of both patients you will note here also areas of decreased and increased density of the inner table with a finely porous outer table and scattered nodules of bone over the skull. This diagnostic change is only found in Paget's disease and has been likened to the picture which would result if the kinky hair of the negro were calcified. Note also that the bone thickening takes place toward the outside, resulting in enlargement of the head and no encroachment on the cranial cavity. These pictures also show the characteristic skull deformity, a flattening of the posterior portion, making the frontal bones seem unduly prominent.

This peculiar and uncommon disease of the bones was first adequately described by Sir James Paget in 1876, and since that time there has been very little discovered which has necessitated any change in his original description. Lewin in 1925 made a rather thorough study of our knowledge of Paget's disease and was able to collect only 250 cases from the literature. Several reports since then have raised the number to about 300. In large clinics, it is reported as being found once in every 10,000 to 30,000 cases, which illustrates its rarity.

It is a condition of late middle life in which the bones undergo remarkable changes in their size, shape, and contour, and the appearance of the bones suggests the name originally given the disease by Paget, of osteitis deformans. Its causes are unknown, but probably it has its basis in some defect in calcium metabolism, possibly a result of a disturbance of function of the parathyroid glands. The bones become thickened and roughened and bend easily, giving rise to the characteristic deformities such as the kyphosis of the spine and the bowed legs. Practically any bone in the body may be involved, but the long bones, and particularly the tibiae, are usually first attacked. The skull changes are

the most characteristic and when well developed are pathognomonic.

The symptoms are comparatively insignificant, at least in the early years of the disease. Vague pains are often complained of, together with loss of strength, and the development of the deformities mentioned. In the reported cases, many have first sought a physician complaining of an enlargement of the head, necessitating a larger sized hat.

The differential diagnosis is not always easy, especially when only one long bone is attacked. *Syphilis* is frequently confused with it, but can usually be ruled out by the history and a Wassermann test. *Osteitis fibrosis cystica* or von Recklinghausen's disease of the bones gives a somewhat similar *x*-ray picture, but in this there is much less bone proliferation, and it is almost entirely a disease of young adults. The skull changes in *acromegaly* are quite different, the facial bones being most affected; exactly the opposite of Paget's disease in which the facial bones are never affected. Also, the characteristic deformities of Paget's disease do not occur in the gigantism of acromegaly. *Tuberculosis* and *osteomyelitis* are localized bone lesions with marked general symptoms. *Osteomalacia* has a more rapid course, usually is associated with pregnancy, and the bones are changed by a diffuse rarefaction. *Metastatic carcinoma* is essentially a bone destructive lesion, leaving patchy areas of decreased density and no appreciable bone proliferation. It more closely simulates Paget's disease than any of the other lesions in the pelvic bones and must be differentiated with care.

The lesson learned from these 2 cases is quite apparent. We did not consider the possibility of Paget's disease when the pelvic pictures were first studied and it was not until *x*-rays of the skull were taken that we made the correct diagnosis.

In investigating the recent literature on the subject, I find that we are not alone in having made this mistake for it is a common one, and has been mentioned in several articles in the last few years. But these articles have appeared in radiological journals only. Bumpus, of the Mayo Clinic, in 1922 stated that the "mistake has been made of diagnosing as malignant an old

firm inflammatory prostate because *x*-rays revealed what was supposed to be metastases to the bones. Carman in 1921 stated that the most pathognomonic changes of Paget's disease occur in the skull and appear as a thickening and density of the inner table with a finely porous outer table and scattered nodules of bone over the vault. Therefore, in all cases of suspected carcinoma of the prostate with bone metastases, *x*-rays of the head to rule out Paget's disease are often necessary before a positive diagnosis can be made. When one remembers that about 16 per cent of all cases of prostatic enlargement are carcinoma, and that one third of the prostatic malignancies metastasize to bone, usually the pelvis or spine, it is apparent that Paget's disease, although rare, must not infrequently be considered.

The prognosis in Paget's disease, as to life, is good, but the condition is usually steadily progressive, and the treatment entirely symptomatic.

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AGRANULOCYTOSIS AND ITS SURGICAL ASPECT

HISTORY

THE first reports of a peculiar type of necrotizing tonsillitis characterized by a marked and especially granulocytic leukopenia with fatal end-result were published by Türk in 1907, Strusberg in 1912, and Marchand in 1913. No additional cases were put on record before 1922, when Schultz described a series of similar cases and claimed them as representatives of a new disease entity which he called "agranulocytosis." Friedemann, whose publications of several identical cases followed the report of Schultz, substantiated the findings and conceptions of this author in regard to the special position of the disease for which he used the term "angina agranulocytotica." Since that time numerous reports of cases showing similar symptoms were recorded in the literature under the before mentioned or some similar names as for instance "mucositis necroticans agranulocytica" (Weiss). By far the great majority of cases described originate from Germany and Austria. While during the first years following the publications of Schultz and Friedemann only very few reports from this country were put on record, their number has been so considerably increased during the last three years, that this country takes at the present time second place in the number of cases reported. All other countries contributed so far a relatively small number of cases. Their total number amounts to about 250 cases at the present time. This estimate includes all cases recorded as "agranulocytosis." But we are

well aware of the fact that numerous cases are reported with this diagnosis which deviate in their symptomatology and their clinical and histologic findings more or less considerably from the description given by Schultz in his original and successive papers on this subject. We consider it, however, an impossibility to establish a definite record in this respect as many of the reports are more or less incomplete preventing a fair judgment of them, or they were published in some local medical journal not accessible to us. According to Friedemann there were only 47 doubtless cases of agranulocytosis on record in 1928. None of the publications contains any comment upon the marked variations in frequency of this condition in the different countries. Our survey of the literature did not offer any reason for this discrepancy which we were able to ascribe to the nature of the disease. We are more inclined to believe that these variations are incidental and more a matter of knowledge and correct diagnosis than connected with the etiology of the disease. We are supported in this conception by the fact that the number of observations has apparently increased in the past years in direct proportion to the degree in which the medical profession became acquainted with the existence and symptomatology of this disease.

SYMPTOMATOLOGY

Previous diseases, especially those of the throat, are apparently without any causative significance in regard to specificity, as numerous different diseases are listed in this group. But it has to be noted that some of these patients had weeks or months before they developed agranulocytosis a severe sore throat from which they seemingly recovered. Others had a period of ill health preceding the onset of the disease. They were during this time easily tired, felt weak and suffered from frequent headache or they had even fever of an occult type (Bantz, Hueper, Petri, Blumenthal, and others). Some of the patients received antisyphilitic treatment with arsenic and bismuth preparations (Kastlin, Reyhe, Thoma, Domarus, Landsberg). Finally the coincidence of previous liver and gallbladder diseases with agranulocytosis has to be noted (Schaefer, Aubertin and

Lévy, Peritz, Hueper and O'Connor, Schultz and Jacobowitz, etc.). In four of Schultz's 23 cases gallstones were present, while we found in 3 cases out of 7 gallstones and evidence of cholecystitis. In the majority of the cases the onset is sudden out of full health. The symptoms at onset are high fever of the continual type (100 to 105 F.), a pulse of high rate and bad quality (110–140), general malaise, dyspepsia. Cases with an almost afebrile course were occasionally seen (Reyhe). A sore throat makes its appearance either right at the start or during the first three to four days. In rare cases the angina may be entirely absent and only ulcerative, necrotizing processes may be present in the esophagus (Schultz). A marked dysphagia usually follows soon, sometimes making swallowing almost impossible. As occasional manifestations of the skin herpes labialis, necrotizing changes of the skin especially at places where the skin is exposed to physiologic or pathologic trauma as the normal openings of the body (mouth, anus, vulva, vagina), cheeks, eyelids, skin around venous punctures, etc., moreover erysipeloid erythemas, vesicles, papules have to be listed. These necrotizing processes of the skin as well as those of the mucous membranes stated below have to be regarded as evidence of a low tissue resistance against the action of the normal bacterial flora. Vomiting and pain in the abdomen may occur. Diarrhea is rarely present. During the course of the disease the development of a slight to moderate jaundice is seen in about 50 per cent of the cases. Schultz considers this symptom as pathognomonic, but Lauter claims that it may be absent. The examination of the mouth gives usually the following findings. The tonsils are in the beginning enlarged, reddened, and may show white to yellowish plugs. Sometimes this condition may represent the whole mouth pathology throughout the entire course. But usually a dirty yellow or gray membrane soon covers the tonsils. Upon removal of this coat an ulcerated surface appears. The break down of the tonsillar tissue may not only start from the surface, but also, according to Schultz, in the deeper tonsillar tissue and later perforate to the surface. In the progress of the disease the tonsils may become greenish black in color and gan-

grenous material may slough, leaving only small stumps of tonsils. Similar necrotizing processes may be found on the pillars, uvula, palate, base of tongue, gums, pharynx, larynx, esophagus representing either extensions of the tonsillar alterations or independent processes. Gingivitis is rather commonly present. An offensive, fetid odor is usually noticeable. The tongue is often heavily coated. Hemorrhages from the ulcers or underneath the mucosa of the mouth are rarely seen. There is no hemorrhagic diathesis of any generalized character present. The submaxillary and cervical lymph nodes are usually enlarged and tender. The heart findings are in general normal except for an increased activity and sometimes irregularity. The blood pressure may be lowered. The examination of the lungs is in the beginning, as a rule, negative; later findings resembling those of a bronchopneumonia may be noticed. The palpation of the abdomen gives sometimes a tenderness. The liver and spleen, the latter more often than the former, are frequently somewhat enlarged. The examination of the urine for albumin is in general slightly positive. Hyaline and granular casts, leukocytes, and erythrocytes may be present in the sediment. Ehrlich's test is negative. Urobilin and urobilinogen are present in cases of latent or manifest jaundice. The bacteriological examination of the throat does not give results of any special significance. Besides the common organisms as streptococci, staphylococci, pneumococci, a fusospirillosis is sometimes observed (in 10 per cent of the reported cases). The most important findings are obtained by an examination of the blood. There is always a considerable decrease in the number of the white cells present which becomes more and more marked toward the end (1500 to less than 100 in 1 cmm.). This leukopenia may precede the onset of the angina for several weeks (Schultz). The granulocytic leukocytes decrease first and may finally disappear completely from the blood. Immature forms of this cell type are rarely observed (Domarus), but it remains doubtful according to Schultz, if these cases belong really in the group of the essential agranulocytoses. Besides the granulocytic leukopenia there is also a more or less marked lymphocytic leuko-

penia which follows closely that of the granulocytes. The monocytes are sometimes temporarily somewhat increased in number in the beginning of the disease. The oxydase reaction is always negative for these cells. In cases which showed remissions or "recoveries" the monocytes increased first and then the granulocytes (Ottenheimer), which were partly of an immature type. The red blood picture (erythrocytes and hemoglobin) is either normal or shows only minor changes. A moderate anemia is observed in cases of protracted course. The number of thrombocytes is either normal or increased. Unusually large thrombocytes may be observed. The coagulation time and bleeding time are normal. The Widal and Wassermann tests are negative. Blood cultures are more likely negative than positive (in about 10 per cent). The positive results were in general obtained during the advanced stage of the disease. Various organisms were cultured (hemolytic and nonhemolytic streptococci and staphylococci, Streptococcus viridans, Bacillus pyocyaneus, B. lactici, B. coli).

The course of the disease varies considerably. In the majority of the cases a rather rapid, often even fulminant course is seen. These patients may die in two days to two weeks after the onset. In others remissions are observed and the course is more or less protracted. Friedemann observed this type in three of his 29 cases. In one of our previously reported cases the remission lasted four months followed by a very acute and fatal recurrence. The recovery of the blood picture during remission is usually only to a low normal level, if it is reached at all. In cases ending in apparent cure the recovery of the blood to its normal status took in general several weeks.

PATHOLOGY

Mouth.—The necrotizing processes in this region may reach considerably varying extensions. There may be only a few superficial ulcerations on the tonsils or there are extensive, multiple, deep, gangrenous destructions involving not only the tonsils, but also the pillars, uvula, base of tongue, epiglottis, larynx, pharynx, and esophagus. They are completely or partly covered

by a dirty yellow, gray or greenish-black coat, surrounded by a wine-red-colored zone in some of the cases. Microscopically the bottom of these ulcers is formed by three layers. A necrotic, granular material intermingled with numerous bacteria is found in the uppermost layer. In the next deeper one which may extend into the muscle tissue the tissue is also necrotic, but the cellular outlines are preserved. Streaklike accumulations of bacteria may be present in the intercellular spaces. Hyaline, fibrinous or red thrombi block the vessels in this area. The absence of leukocytes in the clotted blood is striking. In the still deeper layer living and necrotic tissue are alternatingly found. There is a distinct edema present. Smaller and larger accumulations of lymphocytes, plasma cells, and large mononucleated cells may be observed in varying number. Leukocytes are always absent in these infiltrations.

Lungs.—Subpleural hemorrhages are rather frequently observed. A fibrinous exudate may cover the pleura in places where dark red, solid, irregular, small foci are present in the lung. Such areas are rather regularly found and are especially frequent in the lower lobes, which show also hypostatic hyperemia and edema. The capillaries of these solidified foci of lung tissue are hyperemic. The alveoli are filled with erythrocytes intermingled in places with bacteria. The adjacent alveoli contain an albuminous or fibrinous material. The absence of leukocytes in these foci is remarkable. Gangrenous changes may occasionally occur (Hirsch, Horvath).

Heart.—Subepicardial and subendocardial hemorrhages occur.

Digestive Tract.—Ulcerations similar to those found in the mouth are frequently present in some or all parts of this system, especially in the esophagus and intestine, where they are sometimes very extensive. The intestinal lymph follicles are swollen and the mucosa covering Peyer's patches may become ulcerated, simulating typhoid ulcers. The stomach may contain also single or multiple ulcers, but shows more frequently hemorrhages in the mucosa and erosions.

Liver.—This organ is usually somewhat enlarged and shows evidence of cloudy swelling. Microscopically there exist varying

degrees of fatty degeneration, occasionally multiple, small, focal necroses, and in general an increase of Kupffer's cells. The bile capillaries contain frequently bile casts and the liver cell bile pigment. Interstitial lymphocytic infiltrations are sometimes observed. These liver changes are evidently the cause of the jaundice frequently seen.

Spleen.—A swelling of the spleen is common, but usually not considerable. It is dark red and moderately firm, but never soft as in septicemia. The lymph follicles are not prominent on the cut surface. The sinuses are filled with erythrocytes and proliferating reticulo-endothelial cells and lymphoid cells. Oxydase positive cells are in general completely absent or only scantily present. Hemosiderin is often found in large phagocytic cells. The lymph follicles are small, atrophic, especially the germinative centers which are usually only composed of mature lymphocytes. The proliferated reticulo-endothelial cells outnumber the lymphoid cells in the spleen (Aubertin and Lévy). Small anemic infarcts are occasionally seen.

Lymph Nodes.—The submaxillary, cervical, peribronchial, and mesenteric lymph nodes are in general enlarged. They sometimes contain hemorrhages. The microscopical examination reveals an atrophy of the lymph follicles as present in the spleen and a proliferation of the reticulo-endothelial cells.

Kidney.—This organ shows usually the signs of cloudy swelling. Numerous, red, pinhead-sized points are seen underneath the capsule in the cortex representing swollen, hyperemic glomeruli. On the cut surface the cortex and medulla are not well demarcated. The tubular epithelium is in general markedly degenerated or necrotic. The lumina are filled with casts. There are rarely petechial hemorrhages in the renal pelvis.

Bone Marrow.—The marrow in the long bones may be partly composed of red marrow. The bone marrow is mainly made up of lymphoid cells. Erythroblasts and megakaryocytes are present in normal number. Oxydase positive cells are very rarely seen or are completely absent.

Genital Organs.—Ulcerations of the same appearance and histologic structure as described for the other organs are

rather frequently observed on the vulva, vagina, and uterine cervix.

Schnaase recommends the proteolytic test of bone marrow tissue in cases in which the diagnosis of agranulocytosis is not made intra vitam. He takes 0.2 Gm. of bone marrow from a rib, adds 1.8 Gm. physiologic salt solution and then makes dilution of this emulsion with physiologic salt solution starting with a dilution of 1 : 10, 1 : 50, 1 : 100, 1 : 1000, 1 : 1500. Two drops of these dilutions are placed upon a serum plate which may be divided for matter of convenience into eight fields. One field receives undiluted marrow. After twenty-four hours of incubation at 54 to 56 C. the plates are examined for impressions. Agranulocytic bone marrow will not produce any proteolytic defects in the plate, while the bone marrow of all other cases tested (30) by Schnaase caused impressions. The bone marrow must be removed as soon as possible after death as he observed negative results with this test thirty-two hours after death.

REPORT OF CASES

Case I.—Mrs. F. K. (patient of Dr. Barnes), sixty-three years of age, entered Mercy Hospital on April 15, 1929. She complained of diarrhea, vomiting, pain in the epigastric and right hypochondric region. About three weeks ago, after eating warmed-over spinach she had an attack of nausea, vomiting, and diarrhea. This was followed by an attack of severe pain in the epigastrium. The pain travelled later to the right side, but was not referred to the back or shoulder. The pain has a steady character and was not of colicky type. There was a recurrence five days later, but not as severe as the first time. She had typhoid fever when thirty-six years of age and a nervous breakdown five years ago. She had four children. She was never operated on. Her temperature was 97.8 F. and her pulse rate was 80 at admission. An *x*-ray picture of the gallbladder showed several calculi and an infiltrated wall. The diagnosis was: Acute appendicitis and cholelithiasis. An operation for these two conditions was taken into consideration, but its performance was postponed on account of the blood findings. The blood picture

on April 19, 1929 was erythrocytes 4,200,000, hemoglobin 75 per cent, leukocytes 2000, coagulation time 2.25 minutes, neutrophilic leukocytes 32 per cent, lymphocytes 66 per cent, monocytes 2 per cent. The urine showed on that date occasional leukocytes. On May 6, 1929 she complained of pain in the throat and rectum. The temperature was then 105 F. and the pulse rate 116. The throat was treated locally with mercurochrome and ultraviolet rays. Blood examination: Erythrocytes 3,260,000, hemoglobin 70 per cent, leukocytes 1400, neutrophilic leukocytes 2 per cent, lymphocytes 96 per cent, monocytes 2 per cent. A culture of the throat showed staphylococci, ovoid diplococci, streptococci, gram-positive and gram-negative bacilli. Fusiform bacilli and spirilli were present in the smear. On May 8, 1929 diarrhea, loss of consciousness, difficulty in swallowing, temperature 105.8 F. was noted. Blood examination: Erythrocytes 3,000,000, hemoglobin 70 per cent, leukocytes 1000, neutrophilic leukocytes 4, lymphocytes 94, monocytes 2. Death on May 9, 1929. Permission for an autopsy was refused.

Case II.—Mrs. E. B., sixty-nine years old, entered Mercy Hospital on June 25, 1929. She complained of sore throat, dysphagia. Her consciousness was impaired at admission. She became sick suddenly two days before with sore throat. Since several years she was under treatment for chronic nephritis, hypertension and chronic cholecystitis with gallstones. After her entrance she lapsed soon into unconsciousness, was unable to swallow and had a temperature of 106 to 107 F. A blood examination showed hemoglobin 85 per cent, leukocytes 2200, lymphocytes 100 per cent. Death, June 26, 1929.

Autopsy Report.—Body of a white-haired, well-nourished woman, who appears to be older than her age indicates. The body is extremely warm. Incision in midline from the upper end of the sternum to the symphysis. Peritoneum smooth and glistening. Diaphragm: Left fifth rib, right fifth rib.

Chest: Lungs: Pleuras are smooth and glistening. The right lung is adherent at the apex. Both lungs are well distended with air and show in places small and large emphysematous areas.

In both lower lobes the posterior, dependent portions are almost completely collapsed and of a dark red color and flabby consistency. The other parts of the lungs are reddish gray with a network of black pigmentations. The bronchi contain a small amount of foamy mucus. The vessels are free.

Heart: The left ventricle is firmly contracted. The muscle appears to be slightly brownish red. The muscle is without any scars. The valves are normal with exception of a few yellow patches at the base. The coronary arteries contain a moderate number of these yellow flat elevations. The pericardium is normal.

Aorta: The aorta has a moderate elasticity and shows in its entire length small yellow elevated patches especially around the openings of the intercostal arteries.

Neck: Tongue: At the base of the tongue two bright red, verrucous ulcerations are present on both sides of the midline.

Tonsil: The right tonsil is almost completely destroyed and appears only as a bean-sized, red, irregular mass.

Larynx: Two shallow ulcers with dirty gray coating are present just below the epiglottis.

Trachea: Normal.

Esophagus: Normal.

Thyroid: The gland is moderately enlarged and shows a distinct follicular structure.

Abdomen: Liver: The organ is moderately enlarged, brown red, with smooth surface and shows on the cut surface a turbid appearance.

Gallbladder: The wall is thin. The content is a brown bile and several small, brown stones. The largest one corresponds in size to a walnut.

Spleen: The organ is considerably enlarged, dark red, rather soft. The lymph follicles appear clearly of the cut surface. The pulp is soft.

Stomach, intestine, pancreas, adrenals: Normal.

Kidneys: The organs are of normal size. The capsule strips easily. The surface is reddish gray and smooth. On the cut

surface a turbid appearance and an indistinct medullocortical demarcation is noticed. The renal pelvis is normal.

Urinary bladder: The bladder is distended and contains about 250 cc. of a turbid, brownish urine. The wall is smooth and gray. Urine analysis: Specific gravity 1.016, reaction acid, albumin positive, sediment, a few leukocytes, occasional erythrocytes, few squamous epithelial cells, several coarsely granulated casts, many urates; sugar slight reduction.

Genital organs: The ovaries are firm, white, of bean size. The uterus is small and does not show any pathology, also not the upper portion of the vagina. There is no ulceration of the vagina present.

Microscopical examination: Sections of the tonsils show ulcerative defects of the squamous epithelium. They are covered by a fibrinonecrotic material. In the superficial tissue underneath this coat the lymphoid tissue is atrophic. The vessels contain occasionally hyaline thrombi. Other vessels are filled with erythrocytes in between which large mononucleated, round cells are observed. The follicles of the thyroid are in general of small or moderate size. They are lined by cuboidal epithelium. Occasional lymphocytic infiltrations are seen in the somewhat increased connective tissue stroma. The tubular epithelium of the kidneys is extensively necrotic. The lumina are filled with stringy material. The liver cells show a moderate fatty degeneration. Small focal necroses are occasionally observed. The reticulo-endothelial cells are markedly increased in number. Small lymphocytic infiltrations are present in the interstitial tissue. The follicles of the spleen are small. The sinuses are filled by large round cells and erythrocytes. Leukocytes are not present. Intestine, adrenal, heart muscle are normal. In the lower lobes of the lung areas containing erythrocytes in the alveoli and hyperemic capillaries are seen.

In addition to these 2 cases the authors were called in consultation on 3 cases of which detailed records could not be obtained. Moreover, an additional case was observed recently in this hospital, which will be reported by Dr. Drennan.

GENERAL ASPECTS

Agranulocytosis is observed in persons of middle age, especially in the fifth and sixth decades. But it occurs also in children and young persons as well as in persons past seventy years of age. It is more common in women than in men. The ratio among the sexes varies somewhat according to the different authors (3.5 to 5 women: 1 man). The disease is apparently not contagious. Marked seasonal variations in the frequency of the disease do not seem to exist according to our investigations. But it appears to us that it is somewhat more frequent in spring and fall than in summer and winter.

ETIOLOGY

The etiology of the disease is still essentially dark. But various theoretical conceptions are put forward.

1. Türk and Strusberg regarded their cases as atypical septicemias and assumed the existence of a hypoplastic anlage of the bone marrow as the cause of the unusual reaction of the blood forming organs upon the infection.

2. Schultz considers the agranulocytosis as an infectious disease of unknown origin with a special toxic affinity to the myeloid tissue. He states that it is different from any known and recognized type of septicemia. He points out that streptococcus septicemia has a definite epidemiological factor which is entirely absent in agranulocytosis, that streptococcus septicemia is furthermore mainly a disease of young persons, while agranulocytosis affects in general persons of high middle age and that the occurrence of almost afebrile cases (Reyhe) which were under observation for three months and which died then after a few days of acute illness is not in favor of a septic genesis. Reyhe concurs in this conception of Schultz and emphasizes the fact that agranulocytosis has a uniform disease picture and uniform pathologic findings. He adds that the ulcerations resulting from the low tissue resistance act as introductory foci to secondary infections. He points out also that agranulocytosis cannot be regarded as a very virulent septicemia on account of the cures observed even in some very acute cases (Friedemann).

3. Friedemann asserts that agranulocytosis is the result of an endocrine disturbance (ovary?) which effects an impairment of the transportation of leukocytes from the place of their production into the blood and also of the production itself by the myeloid tissue resulting in a decrease in the number of leukocytes in the blood. All other changes occurring in the course of the disease are according to him of secondary nature. As he refers in his explanation mainly to the ovary as the gland involved, agranulocytoses in men would be excluded from his scheme.

4. Mouzon and Roch and Mozer are inclined to place it into relation to the acute leukemias.

5. The majority of the workers claim that it is an infectious disease and represents a septicemia with atypical reaction of the hematopoietic system (Feer, Weiss, and others) either due to bacteria which possess a specific affinity and toxicity to the granulocytic system (Zadek, David, Sternberg, Pelnar) or due to an atrophy or low vitality of this organ at the presence of a virulent septic infection (Zikowsky). The bacterial cause is according to these investigators either nonspecific (Ehrmann and Preuss) or specific streptococcus (Zikowsky), Bacillus pyocyaneus (Lovett), fusospirillosis (Cannon). Sternberg, Weiss, Pelnar, and others go even a step further and place agranulocytosis in one large group together with leukemias, septic leukocytoses and infectious diseases with lymphocytic and monocytic reactions.

6. Schottmueller asserts that agranulocytosis has neither a uniform clinical picture nor a uniform etiology, but represents a symptom complex.

EXPERIMENTAL INVESTIGATIONS

Numerous authors have attempted to reproduce agranulocytosis in animals by inoculation of bacteria recovered from the blood or from the throat of patients. Different types of bacteria (Streptococci, Staphylococci, Bacillus pyocyaneus, etc., were used and injected into different animals as rabbits, guinea-pigs, and mice, but positive results were not obtained with exception of

the infections with Bacillus pyocyaneus (Lovett) which produced a drop in the number of leukocytes. The injection of patients' blood into animals done by several investigators was unsuccessful. Experiments on rabbits performed by one of us (Hueper) with the above mentioned methods were entirely negative. Roesler and Schittenhelm produced with injections of colloidal substances (dyes, carmin, iron) a temporary or even permanent abacterial agranulocytosis with similar clinical symptoms in animals.

Considering the almost entirely negative results of the experiments on a bacteriotoxic basis these investigations gain special importance as they are apt to throw new light on the causative mechanism of the disease. It is well to remember in this connection the facts, that through repeated injections of colloidal substances a more or less extensive blockade of the reticulo-endothelial system and especially that of the liver is produced, that proliferations of Kupffer's cells, liver cell degenerations, and necroses and inflammatory conditions of the gall-bladder are frequently seen in agranulocytosis, that agranulocytosis as well as cholecystitis are more often present in women than in men, that benzol and arsenic preparations cause liver injuries as well as agranulocytic conditions. It may therefore be well to consider in the future the possibility of primary liver disturbances with secondary toxic effects upon the myeloid system in the etiology of agranulocytosis.

PROGNOSIS

The prognosis of agranulocytosis is bad, but not absolutely hopeless. About 10 per cent of all cases on record were cured (26 cases—Reyhe 2, Lauter 1, Ehrmann and Preuss 1, Zikowsky 2, Starlinger 1, Horvath 1, Leuchtenberger 1, Kommerell 1, Hoche 1, Friedemann 6, Domarus 1, Ottenheimer 1, Schultz 4, Wyott 1, McCall, Gray, and Hodges 1, Blanton 1). The prognosis depends apparently largely upon the extent of the injury to the bone marrow. Friedemann considers cases with tonsillar manifestations as more favorable than those without them. But the tendency of the disease to remissions and recurrences

makes its final outlook always somewhat dubious, as the possibility of a fatal recurrence in some distant future cannot be excluded. Reports of "cures" have therefore to be considered with due reserve.

DIFFERENTIAL DIAGNOSIS

Numerous diseases may resemble in one or the other respect an agranulocytosis. They may be grouped as follows:

1. Diseases with an agranulocytic symptom complex and necrotizing processes in the mouth.

(*a*) Influenza and typhoid fever which in very acute and severe cases show besides a leukopenia with relative lymphocytosis ulcerative processes in the pharynx and larynx differ from the essential agranulocytosis in their course, bacteriological and pathologic findings (noma).

(*b*) Septicemias with atypical blood reaction can be distinguished from agranulocytosis by the presence of a generalized hemorrhagic diathesis, abscesses, primary infectious focus, immature forms of granulocytes in the blood, bone marrow, and lymphatic organs, secondary anemia, frequently positive blood culture, fat marrow in the long bones.

(*c*) Acute leukopenic leukemias and aleukemic leukemias can be differentiated from agranulocytosis by the existence of a generalized hemorrhagic diathesis, secondary anemia, thrombopenia, lengthened bleeding time, positive Rumpell-Leed's phenomenon, tenderness of lower part of sternum, characteristic pathologic alterations as leukemic infiltrations in liver, spleen, kidney, etc.

(*d*) Aleukia (Ehrlich) is also characterized by a generalized hemorrhagic diathesis, thrombopenia, secondary anemia, lengthened bleeding time as some of the before mentioned diseases. It shows as the agranulocytosis an atrophy of the total myeloid system but with inclusion of the erythropoietic system. Moreover, there are always myeloblasts still present in the atrophic bone marrow.

2. Diseases with an agranulocytic symptom complex but without gangrenous ulcers in the mouth.

To this group belong various diseases as the abdominal type of Hodgkin's disease, pernicious anemia, carcinosis of bone, miliary tuberculosis, kala-azar, poisonings with arsenic compounds (arsphenamine), benzol, roentgen rays (especially in the treatment of leukemias), thorium-X.

3. Diseases with similar necrotizing processes in the mouth, but without an agranulocytic symptom complex.

(*a*) Diphtheria, especially the fulminant type, resembles in its general and local symptoms sometimes very closely those present in agranulocytosis, but it is different in hematological, bacteriological, and pathologic respects. In seven of the reviewed cases it was stated that the preliminary diagnosis was diphtheria and in 2 cases of agranulocytosis diphtheria bacilli were cultured from the throat.

(*b*) Vincent's angina may show also locally similar alterations as those found in agranulocytosis as it may agree with this disease by the presence of a fusospirillosis. But the correct diagnosis rests upon the general condition of the patient, the blood findings, the good prognosis, and the pathologic alterations.

(*c*) Monocytic angina (Schultz) occurs as the Vincent's angina at an earlier age (twelve to twenty-seven) than the agranulocytic angina. The necroses are restricted to the tonsils. There is a generalized marked enlargement of the lymph nodes, liver, and spleen present. The latter remains persistent for several weeks or even months after recovery. The blood shows an increased number of leukocytes with an absolute increase of the monocytes (6 to 76 per cent). There is never jaundice present nor are hemorrhages observed. It is apparently contagious (Schittenhelm) and has a protracted course. The prognosis is good.

THERAPY

The therapeutical procedures can be classified into two main groups, those of medical and those of surgical nature.

(*A*) Medical therapeutical procedures:

1. Antiseptics of different kind were locally and intravenously used without any appreciable success. A list of these substances includes urotropin, alcohol, mercurochrome, various silver salts,

and others. The results obtained with salvarsan were not uniform. While good results were reported by Domarus, Schultz, Horvath, Zikowsky, Bantz, the majority of the authors did not see any effect at all from its use. Moreover, it must be stated that other therapeutical means were usually simultaneously employed, a fact which makes any fair judgment in regard to the value of this medication impossible. But it is well to consider the fact that the indiscriminating use of salvarsan may result in an aggravation of the agranulocytic condition.

2. Streptococcus serum, diphtheria antitoxin, parenteral protein, and intramuscular blood injections were also used with varying results. As any specific effect upon the disease agent is out of the question this medication can only act as a stimulant to the bone marrow and the vegetative nervous system. A similar mechanism may have been active in those cases in which temporary remissions or recoveries were seen after the development of abscesses due to secondary infections. We know of two instances in which recovery was obtained after abscess formation. These cases are however not included in our list, as we do not possess their records. Zikowsky believes that pus production may help to break the functional paresis of the bone marrow as, according to him, not an actual destruction of the bone marrow is present, but an inability of the bone marrow to produce and release leukocytes.

3. Stimulating roentgen-ray irradiation over the long bones was successfully applied by Friedemann, Kommerell, Starlinger, Schultz, but was used in many other cases including some of our own without any effect.

(*B*) Surgical therapeutical procedures:

1. Blood transfusions represent the only surgical means so far used in the therapy of this disease. The results obtained with this method are also varying. Some authors report good results and recommend them highly (Reyhe, Schultz, Hoche, Bantz) and advise to make early, repeated and large blood transfusions, while others pronounce them as useless (Zikowsky, Nothmann, Pfab, Friedemann). Schultz comments in regard to the efficacy of blood transfusions that he doubts, if they have any deciding

effect upon the course. We believe that this type of therapy has not received a fair trial as in many instances the transfusions were either given too late or they were not given in sufficient amounts and numbers. A good effect can only be expected from them, if the bone marrow receives a period of rest by the early and repeated administration of large amounts of blood. Moreover, one has to consider that any kind of therapy will probably be useless in the fulminant type of the disease.

2. Finally we wish to propose as a second surgical procedure the drainage of the gallbladder in all those cases which have a definite gallbladder history. Considering the unsatisfactory results obtained with the above listed therapeutical procedures, the frequent coincidence of pathologic gallbladder and liver conditions with agranulocytosis pointing to a possible etiologic connection between these diseases the drainage of the gallbladder may result in the removal of toxic products of causative nature.

CONCLUSIONS

1. Agranulocytosis is characterized by a marked leukopenia giving evidence of an impairment of the myeloid as well as of the lymphoid system. This symptom may precede all others for days and weeks.

2. Ulcerative necroses of the skin and mucous membranes have to be regarded as secondary changes resulting from an extremely low tissue resistance against traumatic factors of various nature.

3. Tonsillar changes may be entirely absent and are apparently not pathognomonic of the disease.

4. The occurrence of long remissions in the course of agranulocytosis calls for caution in the report of "cured cases." Such patients should be kept under observation for many months after their apparent recovery.

5. Agranulocytosis is evidently not as rare as usually suspected, because 11 cases came to our observation during the last three years. It is advisable to make blood counts on all adult patients with a severe angina.

6. None of the therapeutical procedures employed has given

so far satisfactory results in a larger number of cases. Spontaneous recoveries without any medication except of local application of disinfectants have been observed.

7. In about 25 per cent of the cases pathologic conditions of the gallbladder and liver were found.

8. It is suggested to drain the gallbladder in all cases which give evidence of cholecystitis.

BIBLIOGRAPHY

Allen: Ann. Int. Med., 2, 542, 1928.

Aubertin and Lévy: Arch. d. mal du coeur, 21, 369, 1928. (See here also Polish, Bohemian and Skandinavian literature). Bull. et mém. Soc. med. d. hôp. d. Paris, 52, 456, 1928.

Baltzer: Virchow's Arch. f. pathol. Anat., 262, 681, 1926.

Bantz: Münch. med. Wochenschr., 72, 1200, 1925.

Bix: Wien. klin. Wochenschr., 41, 1185, 1928.

Blanton: Jour. Amer. Med. Assoc., 92, 2099, 1929.

Blumenthal: Deut. med. Wochenschr., 54, 1089, 1928.

Borchers: Zeitschr. f. Laryng. Rhinol., 15, 386, 1927.

Botter: Lancet, 1, 440, 1929.

Bromberg and Murphy: Jour. Amer. Med. Assoc., 92, 1266, 1929.

Brüggemann: Zeitschr. f. Hals-, Nasen-u. Ohrenh., 15, 187, 1926.

Campbell: Trans. Am. Proctol. Soc., 28, 11, 1927.

Chiary and Redlich: Wien. klin. Wochenschr., 39, 1510, 1926.

Coll, Gray, and Hodges: Amer. Jour. Roentg., 20, 550, 1928.

Connor: Southern Med. Jour., 20, 141, 1927.

Dahlen and Wahlgren: Acta med. Scand., 65, 407, 1927.

David: Med. Klinik, 21, 1229, 1925.

v. Domarus: Klin. Wochenschr., 8, 779, 1929.

Dosse: Jour. Amer. Med. Assoc., 91, 1718, 1928.

Dufourt: Bull. Soc. de pédiat. de Paris, 25, 509, 1927. Lyon Méd., 141 679, 1928.

Dwyer and Hellwig: Amer. Jour. Dis. Child., 35, 1041, 1928.

Ehrmann and Preuss: Klin. Wochenschr., 4, 267, 1925.

Elkeles: Med. Klinik, 20, 1614, 1924.

Feer: Schweiz. med. Wochenschr., 56, 551, 1926.

Finnigan: Jour. Missouri Med. Assoc., 24, 258, 1927.

Flandin: Bull. et mém. Soc. med. d. hôp. de Paris, 50, 1459, 1926.

Friedemann: Zeitschr. f. Hals-, Nasen-u. Ohrenh., 13, 473, 1926. Deut. med. Wochenschr., 53, 2193, 1926. Med. Klinik, 19, 1357, 1923. Zeitschr. f. klin. Med., 108, 54, 1928.

Gundrun: Arch. Int. Med., 41, 343, 1928.

Haverkamp: Nederl. Tyidschr. v. Geneesk., 2, 2158, 1927.

Hirsch: Münch. med. Wochenschr., 74, 762, 1927.

Hoche: Wien. klin. Wochenschr., 41, 344, 1928.

v. Horvath: Klin. Wochenschr., 7, 1534, 1928.
Hueper: Arch. Int. Med., 42, 893, 1928. Jour. Lab. and Clin. Med., 14, 817, 1929. Hueper and O'Connor: Laryngoscope, 38, 679, 1928.
Jackson: Trans. Chicago Pathol. Soc., 12, 360, 1927.
Jacobi: Wien. med. Wochenschr., 78, 830, 1928.
Jaffé: Münch. med. Wochenschr., 73, 2012, 1926.
Jakobi: Klin. Wochenschr., 7, 870, 1928.
Jedlicka: Sang, 2, 10, 1928.
Koehler: Deut. Arch. f. klin. Med., 155, 155, 1927.
Kommerell: Klin. Wochenschr., 8, 1430, 1929.
Landsberg: Klin. Wochenschr., 8, 507, 1929.
Lauter: Klin. Wochenschr., 2, 1324, 1924.
Lenhartz: Deut. Arch. f. klin. Med., 159, 13, 1928.
Leuchtenberger: Klin. Wochenschr., 8, 1430, 1929, and 5, 1492, 1926.
Licht and Hartmann: Deut. med. Wochenschr., 51, 1518, 1925.
Linthicum: Ann. Otol., Rhinol., and Laryng., 36, 1093, 1927.
Mouzon: Presse méd., 34, 1269, 1926.
Murphy: Northwest Med., 27, 346, 1928.
Nothmann: Klin. Wochenschr., 7, 2159, 1928.
Ottenheimer: Med. Klinik., 24, 1236, 1928.
Paroulek: Arch. d. mal. du coeur, 20, 648, 1927.
Peritz: Zentralbl. f. Chir., 54, 2129, 1927.
Petri: Deut. med. Wochenschr., 50, 1017, 1924.
Pfab: Wien. klin. Wochenschr., 38, 1302, 1925.
Potts: Arch. Otology, 9, 256, 1929. Trans. Amer. Laryng., Rhinol., and Otol. Soc., 34, 90, 1928.
Prendergast: Canad. Med. Assoc. Jour., 17, 446, 1927.
Reiche: Med. Klinik, 7, 2342, 1928.
Reyhe: Klin. Wochenschr., 5, 1059, 1926. Deut. med. Wochenschr., 52, 1153, 1926. Klin. Wochenschr., 8, 1557, 1929 and 5, 1492, 1926.
Roch and Mozer: Presse méd., 34, 1171, 1926.
Rose and Hooser: Arch. Int. Med., 43, 533, 1929.
Rosler: Wien. med. Wochenschr., 77, 599, 1927.
Rotter: Klin. Wochenschr., 5, 773, 1926. Virchow's Arch. f. Pathol. Anat., 258, 17, 1925.
Sachs: Nebraska Med. Jour., 13, 81, 1928.
Schaefer: Deut. Arch. f. klin. Med., 151, 191, 1926.
Schnaase: Klin. Wochenschr., 7, 2342, 1928.
Schultz: Deut. med. Wochenschr., 48, 1495, 1922 and 53, 1213, 1927. Klin. Wochenschr., 6, 2437, 1927 and 8, 1530, 1929. Münch. med. Wochenschr., 75, 1667, 1928. Schultz and Mirisch, Virchow's Arch. f. pathol Anat., 264, 760, 1927.
Starlinger: Klin. Wochenschr., 7, 866, 1928.
Stern and Hartmann: Klin. Wochenschr., 7, 1230, 1928.
v. Torday: Klin. Wochenschr., 6, 923, 1927.
Thoma: Med. Klinik, 24, 2010, 1928.
Thompson: Laryngoscope, 38, 395, 1928.
Trace: Illinois Med. Jour., 55, 36, 1929.

Tschistowitsch: Deut. Arch. f. klin. Med., 162, 231, 1928.
Türk: Wien. klin. Wochenschr., 157, 1907.
Uren: Trans. Amer. Laryng., Rhinol., and Otol. Soc., 34, 516, 1928.
Vos: Nederl. Tyidschr. v. Geneesk., 2, 1474, 1927.
Vos and Staal: Nederl. Tyidschr. v. Geneesk., 1, 2983, 1927.
Weiss: Wien. Arch. f. inn. Med., 14, 303, 1927.
Whitehead: Virginia Med. Monthly, 54, 761, 1928.
Wilson: Amer. Jour. Med. Sci., 77, 88, 1929.
Wyott: New England Jour. Med., 199, 525, 1928.
Zadek: Med. Klinik, 21, 688, 1925.
Zetterquist: Acta med. Scand., 67, 172, 1927.
Zikowsky: Wien. med. Wochenschr., 77, 587, 1927. Wien. klin. Wochenschr. 40, 1376 and 1420, 1927 and 41, 1044, 1928.